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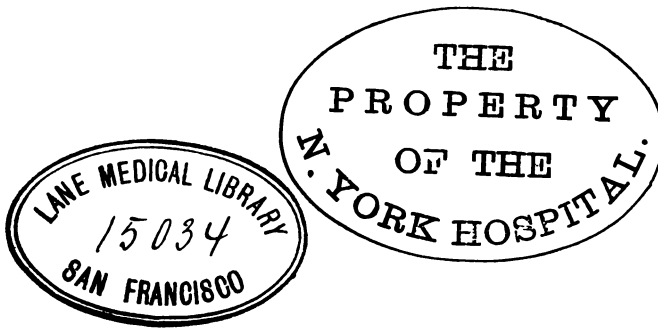
BY

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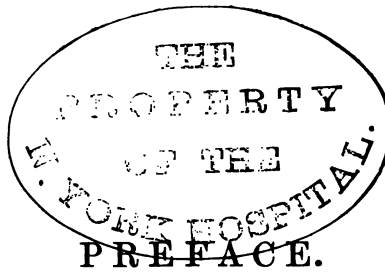
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My original intention was merely to publish examples of the cases upon which was founded my article in the *Journal of Mental Science* for April, 1878. It then seemed to be desirable to preface these by a brief description of general paralysis. But in the course of transcribing from the first rough sketch it was found that the work would far overpass the limits intended, and, in order to curtail it, the very numerous references in the seventh and succeeding chapters were omitted; as also those in the third chapter, which had not been inserted at the moment of transcription. Also, a number of the illustrative cases, at the end of the volume, were omitted, and the rest were very much abbreviated. Yet it is hoped that those retained are sufficient in number and in detail to give some idea of the material upon which was based my description of the several varieties of general paralysis of the insane in the article above referred to.

This work was written in 1878, but other engagements intervened and prevented the completion of the final MS. I have, therefore, added references to a few more recent papers, and to Voisin's systematic treatise on the subject; all of which have appeared in the earlier parts of 1879.

W. J. M.

GROVE HALL ASYLUM, LONDON,
September, 1879.



GENERAL PARALYSIS OF THE INSANE.

PART I.

CHAPTER I.

SYNONYMS — DEFINITION — HISTORY OF DISCOVERY — PRO-
DROMES — STAGES.

Synonyms.—General paralysis of the insane. Progressive general paralysis. General paralysis. General paresis. Paralytic dementia, or dementia paralytica.

(The last name (paralytic dementia, or dementia paralytica), however, should be avoided in practice, inasmuch as it has been employed in at least four different senses: (1) as synonymous with general paralysis of the insane throughout the whole range and duration of that affection: (2) as corresponding only to the later stages of "general paralysis,"—those in which dementia becomes very marked or extreme: (3) as one clinical variety of "general paralysis": and, (4) as descriptive of a group of cases not at all within the limits of true "general paralysis of the insane," but which are instances of dementia with ordinary paralyses, such as hemiplegia.)

"Allgemeine progressive Paralyse der Irren." "Arachnite chronique," Bayle, 1822; "Meningite chronique," Bayle, 1825 and 1826 (the principal motor symptom of which he named "paralysie générale et incomplète."*) "Paralysie générale incomplète," Delaye, 1824. "Paralysie générale des aliénés,"

* A. L. J. Bayle, "Traité des Maladies du Cerveau," etc., 1826, pp. 538, 542, etc.

Calmeil, 1826; "Périencéphalite chronique diffuse," Calmeil, 1859. "Paralysie générale progressive," Requin, Lunier, Brierre de Boismont. "Paralysie progressive, Polyparésie, Démence paralytique. Folie paralytique," Parchappe, 1838-1841, Jules Falret, 1853. "Anoia paralytica" (Fischer). "Dementia paralytica" (Kjellberg).

Definition.—General paralysis is a disease of the nervous system, especially of the brain, marked clinically by (I.) certain general disorders of motility, viz.: ataxy, and, finally, paresis, usually following a certain order and course of development, and especially obvious in the apparatus of speech and of locomotion; also, but in less degree, by (II.) sensory disorder or defect; and marked also by (III.) mental symptoms, which invariably tend to dementia, but in the earlier stages often consist in part of exaltation of feeling, or even expansive delirium. Fourthly, it is evidenced by (IV.) certain organic changes in the encephalon and its tunics, often in the spinal cord and membranes also, and, as some say, in certain sympathetic ganglia as well.

With this definition it necessarily follows that we adhere to the "doctrine of unity" in "general paralysis." Yet on the other hand many, especially among the earlier writers, have deemed it to be merely a complication, or some even a termination of insanity. Of these mention may be made of Pinel, 1812; Esquirol, 1814; Jacquelin Dubuisson, 1816; Georget, 1820; Delaye, 1824; Calmeil, 1826; Foville, 1829; Parchappe, 1836; and more recently Griesinger and Billod, Bucknill and Tuke, all of whom viewed general paralysis in accordance with the terms of the "doctrine of duality."

History of the Discovery of General Paralysis.—The limits of space prevent more than a passing reference here to the history of the discovery of general paralysis. Suffice it to say that, perhaps, the light of a first, yet doubtful, recognition of the disease gleams in the pages of Willis (1672); that Haslam (1793-1809) caught flashes and transient sparkles of the truth in his recognition of certain features of the affection; that Esquirol and Georget did the same with relation to certain other, but more equivocal, features thereof; and that the discovery burst forth with full effulgence in the works of Bayle (1822, 1825, 1826), upon whose heels Calmeil closely trod in this inquiry. Perfect in 1787, and Guislain in his work of 1826, had not recognized the affection.

The Mode of Commencement.—More often gradual in its onset, general paralysis is stated, and particularly by the older writers, to begin frequently by sudden symptoms of cerebral congestion, or by an acute maniacal attack. This I have occa-

sionally seen. The more frequent modes of commencement are detailed below when treating of the symptoms of the first period.

Stages of General Paralysis.—Sometimes three, sometimes four, stages are described. The division into four stages is that of:—First, a stage of mental alteration:—Second, a stage of decided mental alienation, often with active mental symptoms:—Third, a stage of chronic mental disorder or failure, during part of which there is generally a remission of at least the active mental symptoms of the preceding stage, and:—Fourth, the stage in which mental weakness has advanced through confirmed dementia towards amentia, and in which, also, the primary ataxy and subsequent paresis have given place to a general complete helplessness with impaired sensibility, and in which, therefore, there is virtually a complete prostration of the mental and motor powers, and, we may add, of the nutritive, also.

Or a division into three stages may be made, either by omitting the last one, of those just mentioned, as a separate stadium and joining it to the third; or, by omitting the first as a distinct stage, and either joining it to the second, or excluding it as being in reality prodromal in nature.

Such are the stages; but the majority of the cases diverge more or less considerably from this type; any one stage may be absent, and in some no distinct stages whatever can be traced;—a gradually increasing ataxy and paresis, with progressive dementia—or with early exaltation, and later on with dementia,—being the chief outlines.

An example of the classical form of general paralysis may be described as running its full course in four stadia, of which one precedes the decided mental alienation, but inasmuch as three stages are more commonly described, the first of which commences with the first moment of decided mental alienation, it is advisable, with the view of obviating confusion, here to describe four *periods* in general paralysis; the first of which is sometimes termed prodromic, corresponds in the psychical sphere with a mere mental alteration, yet issues from the earlier stages of evolution of that same morbid process which eventually operates a lethal effect upon the physical and psychical powers:—and the last three of which periods correspond to three *stages* of the *confirmed* disease.

The Prodromes.—Under the heading of prodromes are often placed symptoms and signs that really aid to constitute the first period of general paralysis. Yet in certain cases some of these so long antedate the affection that they may be viewed as prodromal. To these, and to others, is reference now made, and they are of the most varied kind.

Some embarrassment of speech is occasionally the very first sign noticed, and Austin, on the authority of Phillips, speaks of extreme contraction of the pupils, and, on his own authority,* of a fixed or unsymmetrical condition of the pupils, as being frequently prodromal; whereas Griesinger† observes that the pupils are sometimes irregular for years before the onset of the disease; while trembling of the limbs‡ has been placed in the same heralding category.

Sometimes the prodromata consist of seizures of cerebral congestion; § or of mere palpitation, flushing and heat of the face and head, with aural tinnitus; or of convulsive seizures of an obscure nature; or of "stunnings," or "absences," especially occurring after excitement or overwork; or of severe headache, or various neuralgiæ, and these last are often severe, protracted, and afflict one part of the body or several parts in succession. Verga|| mentions headache, epistaxis, heat of head, redness of face, somnolence, formication, convulsive, apoplecticiform, or syn-copal, attacks, while later on there may be change of character or exaggeration in everything the patient says or does. Among the prodromata have also been mentioned¶ pto-sis, diplopia, facial palsy, impaired hearing or sudden or transitory deafness; or amaurosis, appearing and disappearing for several years before the incidence of general paralysis, or showing itself anew two or three months prior to the first symptoms of this disease, either to cease again or to remain persistent.

Thus far we have spoken mainly of somatic prodromes; but in addition, or alone, some mental modification is often observed, and generally consists in some alteration of the affective or moral powers, as shown either in demeanour or action, or on the other hand there may be some exaggeration of a naturally choleric and emotional disposition.

If the purely intellectual powers are involved there is mainly a species of inattention. Repeatedly may the patient evince the same particular forgetfulness or blunder, or the conducting of duty or of business may become irregular, fitful, and ill-judged. There may be an expansive, busy, speculative frame of mind; a

* "A Practical Account of General Paralysis," by Thos. J. Austin. London, 1859, p. 65.

† "Mental Pathology and Therapeutics." New Sydenham Society's Trans., 1867.

‡ L. Lunier, *Annales Médico-Psychologiques*, Jan. 1849.

§ In 60 per cent., "*Annales d'Hygiène*," 1860, p. 428.

|| Abstract, *Journal of Mental Science*, April, 1873, p. 158.

¶ *Annales Médico-Psychologiques*, 1859, p. 295, *et seq.*; "*Annales d'Hygiène*," 1860, p. 430.

restless, fitful, yet energetic application to business, though perhaps with some loss of the usual foresight and acumen; a rashness of action, and brusque manner, and a forced and noisy laugh, with unnatural loudness of conversation. To these warnings may be added that of the danger-signal, insomnia.

Or the patient may be depressed, dull, worried about trifles, yet angry if opposed: or full of vague fears and hypochondriacal fancies; and, indeed, languor, ennui and melancholy have been viewed by some* as the most frequent prodromes, the patients having the sad consciousness of their state.

Or, again, with forgetfulness and inattention to duties there may be progressive mental confusion and stupidity, with or without drowsiness and heaviness of head. Memory fails, especially for recent events and recent mental acquisitions, and the power of attention progressively lessens.

Thus, conditions often called prodromal may really come to constitute part of the first period of the developed disease, the transition from the one to the other being imperceptible. Much of the present description applies, therefore, to the first period as well.

Loss of the genital faculties and desire has been observed as a prodromic symptom in some cases.† On the other hand sexual excess sometimes coincides with the development of the first germs of the disease, and when deemed to be a cause is often only a prodromic symptom in reality.

Excess in eating and drinking may now astonish the patient's friends.

But of all the prodromes perversion of the moral sense is the most important. Brierre de Boismont ‡ drew special attention to perversions of the moral and affective faculties in the prodromal period of general paralysis. Even several years (six or seven) beforehand there may be unwonted acts of indelicacy, impropriety, or debauchery, and with these there may be a placid apathy and an utter indolence. He also states § that great irritability may occur two or three years before the outbreak, or menace of suicide, or failure of the usual determination of character, as well as the

* As J. A. G. Doutrebente, Thèse, 1870. "Recherches sur la Par. Gén. Progressive."

† Baillarger, *Gazette des Hôpitaux*, Juillet 16, 1846, No. 83, T. viii. 2nd série, p. 329.

‡ Société de Médecine de Paris, Fév. 20, 1846; *Revue Médicale*, Avr. 1, 1846, T. i. p. 617, and Dec. 1846, T. iii. p. 605; *Gazette Médicale*, Mai 22, 1847, p. 391; Acad. des Sciences, séance, Sept. 24, 1860; "Annales d'Hygiène publique et de Médecine Légale," 1860, T. xiv. p. 405; *Annales Médico-Psychologiques*, 1861, p. 88.

§ "Annales d'Hygiène," *loc. cit.*

6 *The Symptoms of General Paralysis of the Insane.*

failure of memory, or of the clearness or precision of judgment, already referred to.

Often, therefore, is this disease to be feared when sudden unforeseen moral falls,—of which theft is one of the most frequent,—occur to those hitherto without reproach. In the history of many a case do we find that some moral or other mental change in the patient has been noticed long before the recognized onset of the disease. And Guislain * confirms this view from his own experience.

CHAPTER II.

THE SYMPTOMS OF GENERAL PARALYSIS.

IN that clinical phase of the disease sometimes designated “simple” the semeiotic phenomena are mainly of two orders—the mental, and the motor—the latter being represented by incoordination, and usually by paresis tending to terminate in helplessness, to which, also, disorder and, finally, impairment or obliteration of the special senses and general sensibility may add themselves.

To the above, one or more phenomena pertaining to a certain group, and characterized by their suddenness and gravity, are superadded in what has been termed the “complicated” form of general paralysis. In that group of phenomena are comprised the various epileptiform seizures, ranging from those which simulate the epileptic *grand mal*, to the quasi-syncopal which are analogous to the *petit mal* of epilepsy; in it also are comprised the apoplectiform and the simple paralytic seizures—all of which so often break in rudely upon the course of general paralysis. They occur in the most varied degrees, and the epileptiform and paralytic vary extremely in extent and situation, in different cases. Most varied have been the attempts to assign them to this or that lesion.

As already mentioned, a division of the course of general paralysis into stages is often made, but being mainly founded upon the mental phenomena there are many cases to which it cannot be applied, so diverse are the order of evolution, the duration, course, and association of the mental symptoms. Thus, at the commencement we may speak of a sort of incubative stage,

* “Leçons Orales sur les Phrénopathies.” Gand, 1852, T. i. p. 325.

or period of mental alteration ; then of a period of decided, and often obtrusive, mental alienation ; next, of a period in which usually occurs a somewhat progressive decline of the mental and physical powers, chequered by more sudden changes and modifications in the domain of both. If another, and fourth, period be described it corresponds with the latter portion of this period in a protracted case. Of the subdivisions of the course of the disease already made to hand this is one of the most convenient, and will be made use of in the following sections. Nevertheless equally exact though far less striking stages than these can in reality be constructed out of the phases through which the motor signs pass. Even at a very early date (1829) had the elder Foville described a broad distinction between two periods of the chief motor troubles,—in the first of which, he said, there is vigour but some tension of movement, and in the second some relaxation or resolution of the same.

Jules Falret * and L.V. Marcé † described four distinct clinical varieties, at the onset of general paralysis ; two in which the physical symptoms predominate, namely the “paralytic,” and “congestive” ; and two in which the mental, namely the “expansive,” and “melancholic.”

First the physical, then the mental, symptoms will be described in each period ; and although the thermometrical, the circulatory, urinary, and other coincident phenomena are semeiotic it will be far more convenient to speak separately, and afterwards, of them, as well as of certain non-essential but frequent complications, such as the epileptiform, apoplectiform, and paralytic seizures.

First period, the period preceding recognized mental alienation, and sometimes termed prodromic.

A. PHYSICAL SYMPTOMS.

(a). *Motor Signs.*—In the earlier periods these generally escape recognition or record, and, indeed, they may be absent. In the first period, corresponding in the psychical sphere to that of mere mental alteration, the motor signs, if present, are for the most part simply ataxic ; movements of the lips, tongue, and sometimes of muscles about the face and eyelids, being accompanied and interrupted by fibrillary tremor or twitching and voluntary movements effected in these several parts being sometimes preceded and followed by less ample involuntary movements

* “Recherches sur la Folie Paralytique,” etc. Thèse. Paris, 1853.

† “Traité pratique des Maladies Mentales.” Paris, 1862.

in the same direction; in which case there is a repetition of the willed, and therefore, also, of the antagonistic, movements, producing a faint quasi-spasmodic action: and this incoordination becomes more marked in the subsequent stages. Much of the ataxic fibrillary twitching, indeed, may be viewed as a lesser degree of this repetition. Hence when the movements involved are those of speech, there results an occasional embarrassment of it, very faint as a rule at this period, only to be detected by close examination, or even altogether masked, so to speak, by the state of motor restlessness and erethism, with loquacity, into which the patient has sometimes fallen. Chiefly is this embarrassment evidenced by an occasional pause or hesitation, followed, maybe, by an emphasized or somewhat explosive utterance of the next syllable, or there is the elision of a syllable, or a slurring over it, without complete elision, or its incomplete, stammering repetition.

Occasionally, even in a state of voluntary rest, the involuntary spasmodic twitching of the lips, face, eyelids, or occipito-frontalis may occur.

The tongue may be protruded in a sudden, jerky, and momentary manner, but even when this is not the case the incoordination may sometimes be recognized in fine irregular contractions of the protruded organ. In some very few cases a slight incoordination of gait, a budding of the tabic form of gait described by Westphal in general paralysis, is to be seen, but as a rule the coarser movements of the limbs, such as the locomotor, are free at this period, and of the patients many can run and dance apparently as well as ever. Yet the finer movements, such as those concerned in writing, may suffer, and occasionally besides omissions, etc., indicative of the mental state, a shaky and erratic caligraphy bears witness to the motor involvement.

The pupils are now in some cases contracted, irregular, sluggish, or unequal.

A general relaxation of the patient's energy is sometimes described, but this pertains rather to the groups in which dementia, hypochondriasis, or melancholia predominate at the first, although even then the ataxic condition is revealed by indications similar to those above-named.

(3). *Sensory Symptoms.*—Severe headache is occasionally observed, and by some the infrequent cephalalgia is spoken of as being profound, vague, or as if like that of contusion. So also may neuralgiæ affect the head, or may affect the spine or the limbs, especially when symptoms of the spinal order are comparatively prominent. Visceral neuralgiæ are sometimes spoken of.

Crozant* asserted that in the prodromic period of the paralysis of the insane, a general, almost complete, cutaneous anæsthesia precedes the disorders of motility, that it is temporary only, and coincides with the acute period or that of the invasion of the malady. The anæsthesia he attributed to the production of a sort of compression of the brain by the initial intra-cranial morbid alteration in general paralysis. But Guislain† rejected de Crozant's attempt to establish insensibility of the skin as a criterion of general paralysis.

A heated state of the head, flushing of the face, or palpitation, may occur from time to time. So also may vertigo, or a momentary sensation of stunning, or ringing whistling or blowing sounds in the ears.

The Mental Symptoms in General Paralysis.—As in the case of the physical signs, so also in that of the mental symptoms, is it difficult to seize upon and limn any types that can be pronounced to be characteristic of the several periods or stages of the disease. One need only briefly indicate, therefore, the phases of mental disorder most frequently observed in each period; premising that the greatest variety, as well as the greatest changeability, is constantly to be met with in each, and that the mental symptoms in a given case may vary almost from day to day, while yet on the whole a progressive march is observed, unless perchance a prolonged truce is conceded by the lethal malady.

In general paralysis of the simplest form there is solely, or chiefly, an increasing dementia. Yet in agreement with Guislain‡ it may be said that usually there are two orders of phenomena here, the permanent and the transitory; the former consisting of the gradual enfeeblement of conception, memory, and all the mental faculties; the latter, of various forms of mental derangement. Truly, he admitted the existence of cases without obvious mental disturbance, but even in these drew attention to the childish manners and the expression of astonishment.

First period, period of mental alteration.

B. MENTAL SYMPTOMS.

In the first period the indications of incipient mental disorder

* "Société de Médecine de Paris," Feb. 26. 1846. *Revue Médicale*, Oct. 1846; *Annales Médico-Psychologiques*, T. ix. 1846, p. 433.

† "Leçons Orales sur les Phrénopathies," par J. Guislain. Gand, 1852. T. i. p. 339.

‡ *Op. cit.* T. i. p. 327.

are often only a continuation and increase of some of those already mentioned among the prodromes. But whether pertaining to this category or not, they are of the most varied description. As well as the intellectual life are the moral and affective usually touched,—nay, their disorder may be so obvious as to appear primary,—the moral powers suffering more notably in one case, the emotional in another. Sometimes a naturally lively and choleric disposition is morbidly exaggerated in this period, but usually it is an *alteration* in the mental condition, in the demeanour and action, that is observed. Gaiety and self-assurance are frequent; and in one group replace a habitual, or a prodromic, dulness or mistrustfulness. Together with an expansive state of feeling there are sleeplessness, restlessness, or a speculative turn displaying itself in ill-considered or absurd projects for the bringing down of golden showers. The restlessness and ill-directed overactivity may take the form of a superabundant, unwonted, philanthropy and generosity, either rudely planned and busily proclaimed, or rashly carried into actual execution. An expansive egoism, an inflated view of the patient's own position, powers, and aptitudes, is often evinced, and blends this period with the next. Nay, further, as this period actually passes over into the next the imagination, gradually freeing itself from the control of reason, may conjure up visionary schemes for the enriching of the multitude, for the ennobling of mankind, and for the regeneration of the race;—all to be effected by the force and supreme energy of the patient himself. There may be incapacity to carry on the usual avocations, or to fix the attention. Temporary absence of mind, forgetfulness of duties, meals, and appointments, and of the consequences of untoward acts, are often predominant features. Sometimes there is relaxation of all energy; on the contrary, there is sometimes an extreme activity, a restless wandering to and fro, and by night as well as by day, a petulant issuing of contradictory orders to subordinates or to family, a loquacity with self-contradiction and furious passion at the slightest opposition. With the wandering habits far too often are there linked a squandering of means, a buying and selling unnecessarily, imprudently, and at great loss, even while the patient is extolling the acuity and cleverness of his own dealings.

Or, he dispenses indiscriminating largesses; or, sometimes, from forgetfulness and mental weakness and disorder, openly and coolly commits theft. This also occurs in the next period of general paralysis. If he steals, the objects taken are often useless and valueless to him. Quietly, and in the most natural

way, does he carry off articles from under the very eyes of the owners, or even seek the assistance of strangers or of the police for that purpose: if stopped, he coolly confesses his act, or says the stolen articles have been given or lent to him, or belong to him, or he contradicts himself; the meanwhile often displaying an utter absence of any feeling of shame. Baillarger mentions three cases, all females. One stole a variety of articles from shops, and, making no use of them, left the tickets still attached. She, as well as the other two, fell into the hands of the police, and thence into prison, before reaching an asylum. In certain cases, however, as in one related by Dr. Maudsley, the theft is skilfully planned, and a disguise assumed. On two occasions did this patient rob fellow-passengers in a railway train at night.

One of my own patients, in full day, had walked off with flowers in pots from the window of a London mansion, and when taxed with the act said the flowers were a gift to him from the owner. Another, wandering hither and thither, brought home at night a collection of linen from despoiled clothes-lines. Several of my patients, non-commissioned officers, while in this stage appropriated, or failed to account for, moneys passing through their hands in virtue of their office; whereupon court-martial and degradation to the ranks precipitated the course of that intellectual and moral defect and disorder—due to organic disease of the brain—of which the primary defalcation had been merely the outward expression.*

Others make silly, objectless assaults, or become destructive; they do not attempt to conceal acts of this kind, or to escape the punishment and inconveniences that swiftly follow such offences, but on the contrary so do them as almost to ensure instant detection.

Some lie and dissimulate (as it were) in the most imperturbable

* For other cases of theft by general paralytics see—Lélut (2 cases), *Annales Médico-Psychologiques*, T. i.; Baillarger (3 cases), *Annales Médico-Psychologiques*, T. v. 1853, p. 479; "Propension au vol chez les Malades atteints d'un premier degré de Paralyse Générale." Parot's Case, *Ibid.* p. 481; Billod (case of "N."), *Ann. Médico-Psychologiques*, 1850, p. 626; Brierre de Boismont, "Annales d'Hygiène publique," etc. 1860, p. 409; A. Saure (4 cases), *Annales Médico-Psychologiques*, 1861, p. 54; Maudsley, "Responsibility in Mental Disease," London, 1874, p. 75, *et seq.*; *Lancet*, Nov. 13th, 1875, p. 693; Wilkie Burman (6 cases), *Journal of Mental Science*, Jan. 1873, p. 536; and (4 other cases), *Journal of Mental Science*, July, 1874, p. 246; a case mentioned by myself, *Journal of Mental Science*, April, 1872, p. 41; Fabre (2 cases), *Ann. Médico-Psych.*, March, 1874, pp. 198 and 207; F. Darde (6 cases), "Du délire des Acts dans la Paralyse Générale," Paris, 1874, pp. 24, 25, *et seq.*, Obs. xi. to xvi., inclusive.

manner, and others are given over to raging eroticism, as remarked by Billod. Guislain speaks of the occasional occurrence of extreme lasciviousness, or of the grossest fæcal self-defilement. I, also, have known the last occur at this early period.

Much that is done, and especially much that is left undone, indicates a grave enfeeblement of mind ; or ideas are acted upon, without reasoning, as they arise in consciousness, or there is action of a semi-conscious kind, such as is exemplified in an irrational and objectless exposure of the person. But, in another group, the failure to distinguish between the things desired in fancy and actual possessions, as well as a certain expansion of the *ego*, lead the patients to appropriate as their own the property of others, as already described. There is, also, no fixity, no tenacity, in the purposes which engage their mind ; the old schemes are abandoned and derelict, and the new may command but a flagging and flickering interest. Neglectful of personal appearance, of regularity in meals, and of cleanliness in eating them, they pass hither and thither in slovenly and incongruous garb.

In a word some or others of the following phenomena are frequently observed :—The patients are restless, agitated, irritable, speculative, volatile, mobile in resolution ; they buy, sell, or give away, without any adequate reason, object, policy, or necessity, and usually at a loss ; even lose articles of their clothing, seek interviews with personages higher in the state, or pester them with correspondence ; for their extraordinary acts, or thefts, offer still more extraordinary, silly, and varying excuses, and occasionally exhibit salacity, or an open and unconcerned self-exposure.

But besides, or instead of, the more or less expansive symptoms just described there may be other, and differently associated, changes in the moral, emotional, and intellectual faculties—in the conduct, demeanour, energy, and outward life generally.

Thus, in one group differing from those already described the prevailing characters are mental confusion, failure of perception, silly childishness, stupidity, forgetfulness, heaviness of head, drowsiness, and incapacity for the usual avocations. If there have been prodromic sensations of “stunning,” and “absences,” they may continue. The forgetfulness and general mental failure, and possibly a sensory failure, may also display themselves in wet or dirty or uncleanly habits. Moreover, outbursts of some excitement, of insubordination, or even of destructiveness, may chequer the progress of the now incipient, slow-creeping dementia.

In still another group the patients are sad, morose, depressed,

confused, forgetful, inattentive, neglectful of their duties or privileges, hypochondriacal or melancholy in feeling, yet perhaps irascible if roused; troubled and distressed about contingencies either trifling or imaginary. By some a longer or shorter period of depression is deemed the rule. I cannot confirm this view.

Occasionally, the principal phenomena now are suspiciousness, avoidance of society, taciturnity, and a miserly disposition.

Or the confirmed disease may be ushered in by an attack of cerebral congestion, varying in intensity from swimming and aching of the head, mental confusion, ringing in the ears, and pricking about the body;—to coma, with, perhaps, paralysis and convulsions. This may be immediately followed by the fully developed second period of the malady, more commonly called the first stage of the *confirmed* disease; or the patient may drag on for a time without fully pronounced mental alienation.

Nor must it be forgotten that an acute maniacal attack may occur at the transition from the first into the second period.

Second period of general paralysis; or FIRST stage of the CONFIRMED disease.

A. PHYSICAL SYMPTOMS.

(α). *Motor*.—In this period the slight motor signs of the first are sometimes highly developed. Great are the differences in the degree of intensity of the several symptoms in different cases, and some may be absent. As so long well known,* maniacal excitement may mask the motor signs, although in the later stages similar maniacal agitation often only serves to heighten the manifest impairment of speech. The following description, therefore, is applicable merely to a kind of imaginary type, or average, of the cases.

The dysphasia as well as the lingual and labial ataxy are usually decided in this, the second, period, usually termed the first stage of the confirmed disease. Naturally, the consonants are the chief difficulty; especially in pronouncing linguals and labials, or in uttering the syllables of a long word, does the patient fail; a stopping, a faint stuttering is observed, as of one somewhat in liquor; with an effort is the word uttered, perhaps loudly; and at the same time may often be seen a tremulous ataxic or convulsive twitching of the upper lip or of the facial muscles, as in one about to weep. There is a stumbling, as it

* A. L. J. Bayle, "Traité des Maladies du Cerveau et de ses Membranes," Paris, 1826, p. 503; Baillarger, *Ann. Médico-Psychologiques*, 1847.

were, in articulating certain words, an occasional lingering pause, a hesitation, a quivering and thickness in speech; now and then with indistinctness or elision of one or more syllables, and oftentimes with an utterance slow and circumspect. The elision of syllables; the complete repetition once or oftener of a syllable; a species of stammering, or again of stuttering; hesitation, or even sudden momentary arrest of speech, are variously combined. The slowness of speech, so frequent in the next period, may be present in this, also. In this stage, too, as well as in the next may the voluntary movements, especially those of the lips and tongue, repeat themselves several times. This has been referred to in the first period. The disorder of speech varies considerably in different cases, and at different times in the same case, and, indeed, may be almost absent. As the lips, or tongue, are relatively the more affected so is the pronunciation of labial, or lingual, consonants relatively the more impaired. Articulate utterance is worse after sharp exercise, and in marked cases speech is attended with tremor or twitching of the labial and facial muscles. At times this tremor or twitch may be unilateral.

There may be fibrillar tremor of the tongue, which may be jerked in and out in a convulsive manner when the patient is desired to show it. In fact the tremulous tongue may be jerkily, or imperfectly, or only momentarily, protruded.

In some cases the patient even now grinds his teeth, or he may champ the jaws and cheeks, and make movements as of mastication.

Inequality of the pupils is frequent. Sometimes present from the onset this, as Baillarger said, is usual only at an advanced period. When present he thought it indicated that the lesion predominated in one hemisphere. The pupils, then, are often unequal, or unequal at times;—often contracted; often more or less sluggish to light; irregular in shape, one, or both, and constantly or intermittently so; the retina is often hyperæmic; the conjunctiva possibly injected, and, later, perhaps, an irregular dilatation of the pupils may occur. But mydriasis has been far less common, at this stage, in my practice than in that of some who have written on the subject.

The eyebrows are often raised, the occipito-frontalis gathered together or twitching, the lower lines of facial expression partly obliterated; and the features rather florid, or showing dilated venules;—or, on the other hand, the skin may become coarse, muddy, greasy, or of parchment-like appearance.

Later, the movements of the hands become somewhat lessened in adroitness and exactitude, and there may even now be a certain

collapse of the general figure. Especially when there is some maniacal agitation may the movements of the extremities still remain strong and free, but on the other hand they usually exhibit a species of incoordination, and sometimes paresis. The handwriting is sometimes shaky and irregular, the letters ill-formed, or separated from one another, and of hieroglyphical character; a word begun is often not finished, or letters or words are omitted without the patient noticing it, or he breaks down after writing a few words. In its defective manual execution the handwriting displays ataxia; in the omission of words or letters, or repetition of words, or erroneous fusion of words, it reveals fundamental intellectual failure. Yet many patients at this period are busy correspondents, and write long letters to their friends, to parliament, or to the throne. Certain of the above characters are shown in Dr. Bacon's* illustrations, but this cacography usually undergoes marked remissions and exacerbations in general paralysis, and is only in a strained sense of the progressively degenerating nature he describes. Dr. Blandford,† also, long ago indicated the diagnostic value of the omission of words and repetition of sentences in writing, in early general paralysis.

Usually the gait is still fairly free, but in other cases it is somewhat awkward, the steps are long and slightly irregular, sharp turning round may be attended with awkwardness and momentary uncertainty, or even with swaying. A decidedly *tabic* gait may be observed, although this is exceptional. Trembling movements of the limbs, varying in degree and frequency, also attest the ataxia. Now the gait, whether in this or in other stages of general paralysis, varies with the relative proportions of ataxy and paresis present. I say, ataxy *and* paresis, for although the latter is perhaps not absolutely essential, as is the former, yet is it almost invariably present. When the special ataxy of the affection is *relatively* predominant the gait is more or less jerky, irregular, the steps are long, the limbs thrown forward, the movements apt to be hurried and made with unnecessary efforts, the lines of march swerving and crooked. On the other hand when paresis *relatively* predominates the gait is slow, heavy, unsteady, and the widely-separated feet readily trip at any inequality or obstacle on the ground. In advanced cases an unsafe tottering follows upon any sudden attempt to turn. This slow and helpless gait, together with the bended, stooping, frame imprint an aspect of premature senility.

* "On the Writing of the Insane," London, 1870, p. 20; and *The Lancet*, July 24, 1869.

† *Medical Times and Gazette*, Nov. 3, 1866, p. 467.

And this, perhaps, is a convenient place to refer to those cases of general paralysis in which the spinal lesions appear simultaneously with the cephalic, or even precede the latter, or in which, although occurring later, they play an unusually effective part. Particularly in the former of these classes do we obtain a history of severe pains, said to be neuralgic or rheumatic, or supposed by the medical attendants to forecast locomotor ataxy. These, perhaps, have been protracted, have led to incapacity for work, and to tedious treatment. The gait of the patient as a rule takes the *tabic* or *ataxiform* character from the very outset, and the impairment of gait and helplessness of the patient are relatively early and prominent. Moreover, to this state true localized paralyses, due to spinal complications, often come to join themselves, and increase the helplessness.

(β). *Vaso-motor Disorders and Cephalic Hyperæmia.*—Attacks of redness of the face and heat of the head may now occur in all degrees, from mere temporary flushing and hyperæmia, to marked congestion of the cephalic region and brain. These may be due to altered vascularity of, and other incipient changes in, the cervical sympathetic, but more often to cerebral causes, and they are more severe in the later stages.

(γ). *Sensory Symptoms.*—Local colour-blindness has been observed by Batty Tuke and others in general paralysis. A gradual diminution of visual power is occasionally observed even thus early, or it may be more sudden and transient, and perhaps due to circulatory changes; rarely is there visual hyperæsthesia. Hallucinations and illusions of sight are not infrequent at this stage. Auditory hallucinations, too, are sometimes revealed, and may be most vivid and almost unceasing. Claus* observed hallucinations or illusions of sight or of hearing, or tactile illusions, in more than one-third of his cases. Auditory hyperæsthesia is occasionally observed. The muscular sense, the organic, and the tactile often undergo a strange perversion, the patients having the most extraordinary sensations as to the position, expansion, shrinking, or flight of their body or limbs.

It is in this stage especially that Aug. Voisin† has indicated the importance of the loss or diminution of the sense of smell. This he found to be an almost constant and persistent symptom, and to affect either both sides or one side alone. My own experience differs from Voisin's as to this. But hallucinations of smell are very rare.

* "Allgemeine Zeitschrift für Psychiatrie," Band xxxv. 5 Heft. Abstract in *London Medical Record*, Jan. 1879, p. 11.

† *L'Union Médicale*, Aug. 4, 1868, p. 180.

Second Period (First Stage)—Mental Symptoms. 17

Cutaneous hyperæsthesia, and hyperæsthesia of cranial nerves other than those already mentioned, may be observed now, but chiefly at an earlier moment, yet anæsthesia, either real, or only apparent and due to vivid mental preoccupation, is not rare. But any prodromic neuralgiæ scarcely ever continue into this stage.

(8). *Less usual Phenomena.*—Othæmatoma may now make its appearance, and so, too, occasionally, in their various degrees may the epileptiform, or apoplectiform, or paralytic, seizures, to be described in a future section. The intercurrent palsies may be narrowly limited, or unilateral, or generalized.

Nor must we omit mention of the occasional occurrence in this stage of a general subsultus and rapid jerking or tremor of the muscular system, somewhat like shivering or rigor, increased by the erect posture and by voluntary movement, not produced by any impression of cold, and not the rigor of any ordinary pyrexia or phlegmasia, but sometimes lasting for days together, and, perhaps, recurring.

B. MENTAL SYMPTOMS, of the *second period*, the period of mental alienation, or *first stage of confirmed* general paralysis.

The second period is sometimes, but rarely in my own experience, ushered in by a "*congestive*" seizure. Yet Bayle described the usual sequence as being:—a seizure of cerebral congestion; ambitious monomania; mania; dementia.

In this period the mental symptoms may be mainly those of,—(1) expansive delirium; or, of (2) acute maniacal excitement; or, of (3) a peculiar hypochondria; or, of (4) a melancholia; or, of (5) a dementia; or, in a few cases of (6) "*stupor*" (of the character either of acute dementia or of *melancholia attonita*); or, finally, of (7) circular insanity.

Yet a certain mental disharmony, confusion, and dementia are of the essence of the mental affection throughout general paralysis, and are analogous, at first, to the ataxy, and, then, to the paresis, in the motor sphere.

It has been said (Salomon) that the distinguishing characteristic of this stage is the confusion, owing to his defect of judgment, that the patient makes between his ideas or beliefs and his desires, so that to him they are the same: or, as Billod* long ago suggested, for him the mirage is such that he believes he possesses that to which he aspires, and holds as realized his dreams of

* *Annales Médico-Psychologiques*, 1850.

happiness.* Esquirol† appears to have particularly noticed the contentment and indifference of the patients, the facile diversion of their attention, and their easy-going acquiescence in their enforced detention.

In describing the several semeiological forms, viewed merely from the psychical side, it would be useless and cumbersome in this place to make, like certain observers, several divisions of each form; as, for example, to make a quadrifid division of the expansive form into:—1. Mania of riches, of grandeurs, predominant and persistent;—2. Exaggeration of the *ego*, contentment, satisfaction, occasional ideas of wealth, of grandeur;—3. Mania of grandeurs, of wealth, only at 'long intervals;—4. Double form, expansive and oppressive, with ideas of wealth and grandeur.‡

Subdivisions of this kind are all the more unnecessary as none of the phenomena are persistent, as they interchange rapidly and frequently, and are in reality but phases of the same morbid mental condition.

(1). *The Form in which Expansive or Ambitious Delirium predominates.*—By many writers extraordinary stress has been laid upon the description of the extravagant delusions and the emotional exaltation of general paralysis, and so striking are these phenomena that attention has been too much withdrawn from other, and equally common, facts pertaining to the mental order of symptoms.

When the exaltation is highly developed we find the patient with an inflated mien, an expansive, smiling, benevolent expression, an impressive bearing of good-fellowship and friendship; overflowing with good-nature, thanks and compliments; pleased with everything and everybody; delighted with, and sounding the praises of, his surroundings, however mean and irksome they may be; loquacious, singing, restlessly moving about, dancing, or capering; eating rapidly and with avidity; covering his hat with some fantastic or tawdry ornaments; destroying his clothing and stitching it together again, to improve it, as he says; or, when the symptoms verge towards, or attain, the maniacal, passing the night in re-arranging his bedding a thousand times, or in tearing it up with the avowed benevolent intention of making a dozen, or an hundred, beds out of the one supplied to him.

With this there may be hallucinations of sight or of hearing,

* But see the section on the "Pathological Physiology of General Paralysis," *infra*.

† "Des Maladies Mentales," par E. Esquirol, Paris, 1838.

‡ See paper by Author, *Journal of Mental Science*, Apr. 1878, p. 27.

in harmony with the extravagant notions. These notions usually run upon the possession of enormous wealth, high titles, prestige, position, great muscular powers or sexual capacity, marriage to members of royal houses, and perhaps to many of them; the distinguishing features of these delusions being that they are multiple, varying, ridiculous, and self-contradictory; and betoken an abrogation of the power of judgment, while they often *culminate* as if in a crescendo movement of the expression of magnificence. The patient is not only "possessed," but inflated, with greatness. The methods of language fail him here as he rides uplifted on the mighty wave of feeling; or, to him borne on this swelling tide of exultation, the very heavens appear to open, and he holds converse with celestial beings, and has ecstatic visions of eternal fields. Last flight of all, he may announce himself enthroned as the Almighty, and invested with His sceptre of universal sway, amid the pæans of angelic hosts.

The exalted delusions announced by him are the indicia of a progressive delirium, which contrasts with the fixed monomania of pride or ambition sometimes seen in ordinary insanity.

To read the descriptions of some writers one would suppose that such as these were the almost constant and abiding mental symptoms of general paralysis. This, however, is rarely the case, even in the expansive form. More often symptoms like the above alternate with hypochondriacal feeling and ideas; or with a whining, moaning expression of peevishness and distress; or with childish and unnecessary fear and terrors; or with sullen irritable states of feeling; or, finally, with a condition of simple dulness, stupidity and confusion.

Frequent, also, in place of pure grandiose optimism, is that condition of quiet, pleased, smiling, self-satisfaction seen in some, but not associated with decisively exalted delusions. These are they who tell with complacency, though without marked exaggeration, of their possessions, however slender the latter may be; dwell upon the looks and personal qualities of their common-place wives or husbands and children; live amidst a few reminiscences of the past flattering to their vanity; but who, if questioned, exhibit none of the above extravagant delusions; though a soft brightness lights up for them the beauties of their now narrow mental world. They are "well" and it goes "well" with them, say they, with hesitating tremulous utterance, breaking health, neglected work, and shattered prospects; thus proclaiming a most striking proof of that utter prostration of perception and of judgment which already announces the imminent ruin of the whole edifice of their mental life.

2. *The Acute Maniacal Form.*—An acute maniacal attack of some duration may occur at the transition from the first to the second period, or, as some would say, from the prodromic to the first stage; or attacks of this kind, but of a more ephemeral duration, may be found throughout the second and third periods. Yet a more persistent condition is now referred to.

In the acute maniacal form, add to much in the foregoing section the symptoms of extreme mental and motor agitation; with insomnia, and restlessness at night as well as by day, and occasionally hallucinations of sight or of hearing, or even illusions of some of the other special senses. An incessant motor activity possesses the man; he talks, shouts, sings, stamps, seizes and destroys surrounding objects, tears his clothes and bedding or soils them, smears himself and his room with his own ordure, or drinks his urine; sometimes even neglects his food, or seizes and hoards pebbles, rags and rubbish as if they were "inestimable stones, unvalued jewels." At times he may be most dangerously hostile, threatening, or violent, and assault others far more powerful than himself. His temperature ranges higher than normal, his eye is quick and bright, and his countenance vividly injected.

The *agitation bruyante* may be succeeded by an *agitation silencieuse*, of the character described below (p. 31), and, in its turn, the latter may again make way for true maniacal excitement; as Falret well observed.

3. *The Hypochondriacal Form.*—With this form the name of Baillarger* is particularly associated. He it was who first declared "délire hypochondriaque" to be a characteristic symptom of general paralysis, saying that as ambitious delirium was that special to excitation in general paralysis, so the hypochondriacal delirium was that special to depression in the same disease. Moreover, that this was a special hypochondriacal delirium, found only in general paralysis. No doubt this is too exclusive a view. According to Baillarger, also, the hypochondriacal delirium was of evil omen in general paralysis; when it was present the tendency to gangrene was enhanced, and occurred at an earlier period than usual; if it was prolonged the patient fell into marasmus. He also deemed the hypochondriacal delirium a valuable precursory sign; for when it was observed in cases classed as melancholia he sometimes found that afterwards the patients were recognized as being the subjects of general paralysis.

A hypochondriacal, or a melancholy, feeling of depression may

* *Gazette des Hôpitaux*, Feb. 3, 1857, p. 55; *Ibid.* May 9, 1857, p. 218; *Ibid.* Oct. 13, 1857, p. 477; *L'Union Médicale*, 1857, p. 385; *Annales Médico-Psychologiques*, T. vi. 1860, p. 509.

occur among the prodromes, or, again, in the first period, that which is sometimes termed prodromic and is antecedent to mental alienation, but the pronounced hypochondria of general paralytics is more apt to occupy part of the second stage of the *confirmed* disease (third period), and less often of the first stage (second period). Yet the mental symptoms may more or less partake of melancholic or hypochondriacal characters throughout, and these may be intermingled. When marked depression occurs at the commencement, and thence is continued into the first stage of the *confirmed* disease, the delusions assume by preference the melancholic form, and in the soldiers under my care are usually of a terrifying nature; the patients, stricken with anxiety and terror, assert that they are persecuted, hunted, insulted, or that they are to be shot, or they beg that they may not be killed. As I* have stated elsewhere, "they are anxious, querulous, worried, and bewildered, distressed by trifles, suspicious of harm, sometimes have hallucinations of sight or hearing, or are either suicidal or hypochondriacal. These symptoms may continue even to the late stages. If occurring for the first time during the middle stages, the hypochondria co-exists with a shattered understanding, and the delusions are usually of the following nature:—The patients say of themselves that they are "dead," "have no head," "have no throat and cannot swallow," "their intestines are gone," "their eyes have been extirpated," "their testicles are wasted," or that they "have been castrated," or that "part of their body is dead." With this there is often a refusal of food and obstinate resistance to feeding, a wild howling when they are interfered with, an inability to walk or stand, a huddling up in bed, and general spasmodic tremor when locomotion is attempted or when they shrink from contact."

Having thus, from my own practice, as just quoted, illustrated certain clinical phases of hypochondriacal delirium in general paralysis, I may now add other cases, from the same, taking the following forms.

In one; after a period of exalted delusions with considerable maniacal agitation, hallucinations, insomnia, and restlessness, the patient for a time is usually silent and of apathetic appearance, but now and then suddenly and paroxysmally breaks forth into the reiterated shout, "Come here my cavalry—my cavalry come here;" or, as if in a paroxysm of terror, "Oh God have mercy on my soul—oh God have mercy on my soul."—Later, the physical signs continue well-marked, and the face muddy, sallow, and

* *British and Foreign Medico-Chirurgical Review*, April, 1877, p. 457.

relaxed, with apparently enlarged and flattened features, which are almost expressionless—except of a certain apathy. Several months later he says, in one breath, that “the world has nearly starved him,” and he “is nearly dead”; and, in the next, that he “is first class,” “is the Crystal Palace,” “is the virgin Mary,” and “is Adam and Eve,”—all this with a dull unhappy look, and weeping soon coming on.—Still later, he sometimes denies any exalted ideas, and at other times, when more than usually quiet, dull, heavy, stupid and feeble, he says, if closely questioned, that he is “nearly dead,” and to almost every question replies “I don’t know.”—Garnishing his words with coarse additions, he says at a still later period that “he is nearly dead”; that “his legs, his wig (he wore none), his chest, and belly are gone to hell”; that “his little eyes are blind,” “his little nose” contains no mucus, his virile organ “is of no use to him”; and that he “can’t take food and will be dead by night-time.” Only a few minutes afterwards he says that he has no money, but adds that he “owns all the world and the regiments belong to him.”—Subsequently, epileptiform seizures took place.

In another case, the patient, after a period during which exalted delusions with some excitement and violence were evinced, became restless and sleepless at night, and more dull, heavy and confused in mind; the face and head became flushed and heated; the gait and various movements of the limbs, as well as of speech, displayed a great increase of ataxic irregularity and trembling. In anxious perturbation the patient strove to tear off his clothing and get under the water-tap under the delusion of imaginary *fæcal* self-defilement and false sensation of *fæcal* lumps about the nates. Saying he was not well—indeed was “very bad and very dirty,”—and that there was nothing to eat, he refused food. This continued; nervous and frightened in appearance, he resisted most strenuously his being fed and all the necessary tendings of his nurses; struggled to get here or there without apparent aim or object; grasped tenaciously, and pulled or pushed at, any surrounding object as the door-handle or the clothing of a bystander.—Later on, trembling as he stood, or as he advanced with tottering, jerky, slow, and apparently feeble steps, he still would raise a shaky hand to push against one, or to seize a hand, grip it hard, and thrust away or pull towards himself its owner; in everything taking the contrary course to that desired of him. Held by one attendant, he was fed by another, the meanwhile bellowing between each mouthful. I might add many other cases to these.

In the hypochondriacal, as in the expansive, form, the

delusions are usually numerous, varying, self-contradictory, and absurd.

Oftentimes a striking feature about the hypochondriacal delirium of general paralysis is its appearance in sequence to the more common expansive delirium, and then, indeed, it often is intermingled, or alternates, with the latter. From this blending arises such extraordinary delusions as some of those just mentioned where the patient, deeming himself at death's door, with obstructed passages and annihilated organs, and without any pecuniary resources, yet, in the same breath, declares himself to be the Crystal Palace, and claims proprietorship of the world.

The tendency to believe the bodily organs or limbs diminished in size, as noticed above in the same case, is not at all uncommon.

The frequent delusions that the mouth, throat, or bowels are closed may induce the patient to refuse food, and enforced alimentation may become necessary.

Very similar to several of those already referred to in my own practice was the following French case*:—At first a period of incoherence and maniacal excitement, followed by the physical signs of general paralysis, or at least by their recognition, and, later on, some notions of grandeur together with dementia,—until at last the patient one day refuses to rise from bed, lets herself fall to the floor, and, speech being free and intelligence not wholly inactive, declares that this or that one, or all, of the bodily organs is, or are, “gone,”—that she “has no blood and no breathing, but is dead and cut to pieces.” The case in mind died after six weeks with gangrenous eschars over the sacrum, toes, and shoulder-blades.

But all the cases of this form are not characterized by the same symptoms; some, corresponding in part, I think, to such as Falret† described under the “depressive” variety, have a melancholic appearance, are apathetic, immobile, speak but little of their own accord, wear an expression of indifference and want of mobility; the features, not concentrated, as in melancholiacs, towards the median line, are as if pendent and without any tension, and the face appears to be enlarged and flattened;—a special facies, somewhat like that of double facial palsy, and expressing silliness and absence of ideas rather than preoccupation; while, occasionally, spasmodic twitches of the face are observed, especially during speech. Speak to them and they

* *L'Union Médicale*, 1857, p. 385; *Gazette des Hôpitaux*, Oct. 13, 1857, p. 477.

† “*Recherches sur la Folie Paralytique*,” etc. Thèse. Paris, 1853.

smile, their countenance expands, and expresses general satisfaction and great feebleness of intellect. Rather than sadness, they exhibit apathy, or even have a vague and general contentment; perhaps say they want for nothing, and often have some ideas which reflect satisfaction, or even certain ideas of grandeur, which sometimes suddenly light up their countenances. This state, as a rule, occupies only one period in the course of general paralysis, and is usually preceded or followed by excitement. Such at least in brief abstract, is Falret's description of a condition which he agrees with Baillarger in assigning more frequently to the female sex.

Patients of the above groups do not often volunteer their statements. Left to themselves, they are generally silent; interfered with, roused, or questioned, they may reveal the absurd hypochondriacal notions; in fact, as noticed by Legrand du Saulle,* the hypochondriacal delirium of general paralytics often must be sought for to be verified. It is, however, extremely doubtful whether the proportion of cases presenting hypochondriacal symptoms is as great as he says (sixteen to one) in paralytic, as compared with non-paralytic, insanity, with depression.

In cases of the hypochondriacal form Baillarger said the prognosis was of the worst, and Jules Falret held that the cases with the "melancholic" form of onset were often short. But in my own experience, while the general results are the same, and chiefly in early typical cases, yet *some* of the cases presenting these marked hypochondriacal symptoms at some part of their course are amongst those of comparatively long duration; and on turning to Austin's work I find that his experience was similar, except in those cases where the "melancholic" delusions (under which he evidently classes the hypochondriacal) were of a very dreadful character.

Already has reference been made to the assertion of Baillarger that there is a gangrenous diathesis, a tendency to easy mortification of the tissues, in these cases. I have seen this in some typical examples, but not to so unusual an extent in certain others where the hypochondria was temporary and intercurrent; nor have I found, like him, the inequality of the pupils markedly greater here than in other cases of general paralysis.

4. *The Melancholic Form.*—The peculiarities of the hypochondriacal delirium in general paralysis stand confessed in the above description.

But other patients, or the same, have the more ordinary melan-

* *Annales Médico-Psychologiques*, 1861.

cholic delusions, depression, sadness, or even suicidal impulses; * some dread or terror of impending evil weighs upon them, and the ideas run on persecution, poisoning, or spiritual perdition. These melancholic delusions have already been briefly referred to at the beginning of the preceding section. A. J. Linas,† indeed, denying Baillarger's views as to the special nature of either its ambitious or hypochondriacal delirium, showed, that in general paralysis, depression might take not only the hypochondriacal form but also assume every shade of depressive delirium, and I may add that cases illustrative of this may be found even in the pages of the earliest writers on the subject. Yet has it not in general paralysis the cohesion and fixity of ordinary melancholia. The delusions of persecution betray too great an incoherence and mutability. If so be that these patients attempt suicide or homicide there is usually no fixity in the reasons they assign; no precaution, no concealment, no persistent aim in the accomplishment of the act itself. There is the same obvious childishness in the suicidal acts or attempts—the same mental, moral, or affective enfeeblement—as in the expansive form.

With the suicidal, there may be also homicidal inclinations or attempts, and that either simultaneously or alternately.

With the melancholic ideas hypochondriacal may also flourish.

In one general paralytic under my care, together with marked mental enfeeblement, there were protracted delusions of annoyance and of injury;—depressed and weeping, he declared that his comrades "were against him."

Another suicidal general paralytic spent his days in fear, alarm and suspicion: and was querulous, restless, anxious, worried, distressed, and unable to engage in any occupation. He evinced visual hallucinations, and also expressed melancholic delusions of a common kind,—asserting that "every one was against him," and that poison was constantly administered to him in his food.

Another patient, admitted to a military hospital with vaguely reported head symptoms, and then with "paralysis," and then discharged to light duty, was readmitted in a state of some "stupor and palsy," with delusions that he was to be hanged or shot, and with suicidal, and homicidal, tendencies. Later on, when under my care, there were depression of spirits, insomnia, incoherence, confusion of ideas, impairment of memory, halluci-

* In 529 cases Castiglioni stated that 80 had made, or had threatened to make, suicidal attempts. Quoted by Dr. T. Harrington Tuke, *Journal of Mental Science*, Oct. 1860, p. 95.

† "Recherches Cliniques sur les Questions les plus Controversées de la Paralyse Générale." Thèse. Paris, 1857. No. 193, p. 35.

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nations of sight and of hearing, delusions that he must either be hanged or be shot, that he was "warned" daily to that effect, and that his mother was in the same hospital. Subsequently were observed epileptiform seizures, many attacks of paralysis, occasional wild and noisy excitement, or self-burial in bed-clothes, as if in terror.

A number like these might be related.

Melancholic delusions of the religious order, or relating to imaginary poverty, or even a melancholia agitata, may be observed.

According to one observer weeping in general paralysis is usually sympathetic with intestinal ailment; according to another, general paralysis from alcoholic excess is specially characterized by visual hallucinations, delusions of persecution and of poisoning, with homicidal and suicidal tendencies.

Several observers, and among them Dr. Wm. Wood,* have noticed the greater relative frequency of the melancholic form in *female* general paralytics. Dr. T. S. Clouston found melancholia more frequently among those general paralytics who were phthisical.

5. *The Form in which Dementia predominates throughout.*—Cases without expansive delirium, excitement or depression may with convenience be grouped separately, and this without any prejudice to the view that clinically the mental basis of general paralysis is essentially a weakness, a pseudo-dementia, or a dementia.

In a few cases of general paralysis a simple dementia begins, progresses, and includes the entire range of mental symptoms throughout the whole course. It is a question whether these are not examples of general paralysis in its pure and simple form. Although embraced under the present heading, these are not more referred to here than are the cases in which dementia, although the predominant symptom, is associated with other mental phenomena, such, indeed, as may occur in the dementia of ordinary insanity.

As I have stated elsewhere,† the most frequent mental symptoms in these cases of general paralysis are as follows:—

"General intellectual weakness and loss of memory, increasing to fatuity if the patient survives long enough, and evinced by blunting of perception, of apprehension, and of reasoning power,

* *British and Foreign Medico-Chirurgical Review*, July, 1860, p. 198. Also Baillarger, Jules Falret, &c.

† *British and Foreign Medico-Chirurgical Review*, April, 1877, pp. 449, 450.

by confusion of thought and of verbal expression, and a slowness and dulness of mental operation, the patient, also, paroxysmally becoming more 'lost.'

"Incoherence of speech.

"Long periods, or paroxysms, in which the patient is restless, meddlesome, prone to self-stripping, destructive to clothing and bedding, or apt to wander about listlessly.

"The habits soon become degraded. In some cases occasional paroxysms of excitement arise; in a few there are delusions of impending danger or of injury, together with terror, in the earlier stage, and at this time the patients may attempt to escape from the vaguely apprehended evil; in a few, also, insomnia, or even hallucination, is noticed, but this last is very rare.

"The emotional display and emotional facility are less marked in this than in any other of the symptomatological forms of general paralysis. Still, at an early period, there may be found a slightly expansive state of feeling, a shadowy gaiety of self-consciousness, which, however, soon disappears; or, on the other hand, an irascibility or petulance on the slightest occasion."

Early abolition of speech with persistence of inarticulate cries is not infrequent in this form.

6. *The Form with "Stupor."*—In a few cases general paralysis sets in with symptoms of acute dementia, or (it is said) of *melancholia with stupor*. Here the ordinary motor and sensory signs of general paralysis are either absent at first, or are masked, as it were;—when the extreme mental symptoms pass off it becomes possible to verify the existence of the physical. But not always; for a marked remission, or apparent recovery, may immediately succeed the acute symptoms, some weakness of the intellectual powers remaining. The ataxic troubles, whether of speech or of other orders, may now slowly become obvious, or, if already present, may increase, and the patient's mental alienation again become patent.

In one such case, afterwards under my care, the patient not returning to his house as usual one day was found at 4 A.M. in the street, unable to give any account of himself, but not under the influence of alcohol, or of any drug. Subsequently he ceased to speak or to take food, and, absolutely silent, sat with bowed head, taking no care of himself, and passing evacuations where he sat. When admitted, no reply could be elicited from him, his habits were still as objectionable as before; to the necessary artificial feeding he opposed a strenuous resistance; when left alone, he sat as if helplessly, with head for the most part hanging down, and eyes gazing in one direction, but no marked terror

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or emotion of any kind limned itself in his features. The pupils were sluggish, the right larger and somewhat dilated, the left small and slightly irregular in shape. The pulse was 114, rather small, and compressible. The viscera were healthy. Later on, diarrhœa was troublesome, but was perhaps connected with the artificial feeding. In about three weeks he began to reply in monosyllables, and after this the mental dulness gradually cleared away, the speech and writing were now noticed as being like those of general paralysis at its commencement, the frame was bent, and the gait slouching and unsoldierly. Finally, he being now only somewhat childish and weakly emotional, the urgent desire of his friends for his discharge was acceded to after five months of treatment. As foretold, before very long he relapsed, but into what phase of mental alienation I am not aware.

7. *The Form with Symptoms of Circular Insanity.*—Dr. Fabre* describes a form of paralytic insanity (general paralysis) characterized by alternate excitement and depression (circular insanity). When there are only two phases ("à double phase") they succeed each other suddenly, while in the form which is of triple phase there is an intervening period of calm, or, (1) excitement, (2) calm, (3) depression; in this differing, therefore, from the non-paralytic circular insanity, in which the usual order is (1) excitement, (2) depression, and (3) period of quietude or lucidity.

I have observed the above clinical form of general paralysis of triple phase.

During the phase of excitement, besides maniacal symptoms, are ideas of contentment and satisfaction, expansive delirium, and sometimes delusions of persecution. During that of depression, there may be melancholia even to profound stupor, melancholic or hypochondriacal delusions, and even a suicidal tendency. But several of Fabre's cases seem to be merely marked instances of the transformation often observed in the psychical sphere in general paralysis, a recurring fluctuation without the distinctness of simple circular insanity. In one case, too, the phase of depression seems to have preceded that of excitement.

Renaudin and Lunier, however, had long ago reported instances of general paralysis with symptoms of circular insanity (à double phase), parietic signs coming on in one case in the period of depression, and in another during a state of chronic mental disorder.† Another observer‡ described a case in which excite-

* *Annales Médico-Psychologiques*, March, 1874, p. 197.

† *Teste Baillarger, Ann. Méd.-Psych.*, 1858, p. 403.

‡ *Brierre de Boismont, Ann. Méd.-Psych.*, 1859, p. 329.

Third Period (Second Stage)—Speech ; Tongue. 29

ment was followed by depression, and this by convalescence and discharge; then, subsequently, there appeared, successively, high exaltation—physical signs of general paralysis—depression—pronounced paralyses—and death. Twelve others mentioned by the same do not appear to be examples of a “circular,” clinical variety of general paralysis, but only of its emotional fickleness and variability.

III. *Third Period of General Paralysis, or SECOND STAGE of the CONFIRMED Disease.*

A. PHYSICAL SYMPTOMS.

(a). *Motor Signs.*

Speech.—In this stage the articulation becomes more imperfect and shaky than before. The words are jumbled together, or there are slowness and stuttering, and an increase of the former hesitation to a momentary arrest of speech, usually ending in an explosive utterance with, perhaps, the elision of syllables. Or, with a mumbling and drawling utterance the voice is thick, hoarse, coarse, or muffled; and the accompanying twitchings of the lips, face, and tongue very marked. Let the patient attempt to speak, and often the antagonists of the muscles which commence to act are called into play: hence interference with, followed by recommencement of, the first movement; and these direct, and opposed, movements not only precede, but also follow, the purposed, and finally accomplished, movement. But the speech is often made slow and deliberate, as if better to surmount the difficulty, while the lips are sometimes brought more closely together, and the gap between the teeth lessened, whence, occasionally, a pendency or a stiffness and immobility of the upper lip. Yet one cannot readily agree with C. Gallopain that the hesitation, and again the slowness, of speech, and approximation of lips are always, or often, purely intentional, and are conscious stratagems on the part of the patient to obviate the lingual and labial ataxy; nor, again, that the final mutism is sometimes sullenly willed because of the annoyance the patient feels at the dysphasia.

Tongue.—As a rule the tongue is now only protruded, at the best, in an imperfect or momentary manner, jerkily and helplessly, moving backward and forward or from side to side as if the sport of varied impulses. The reflex activity and the sensibility of the pharynx and soft palate are lessened, and these parts are often relaxed and congested; the laryngeal sensibility and its muscular energy may become impaired: and, in the later periods, the par-

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ticles of food tend to pass into the larynx; deglutition is also impaired, and attempts to swallow solid food may induce asphyxia.

Pupils.—The pupils have now become sluggish, irregular in shape; and, generally, *unequal* as first described by Baillarger;*—often, one is contracted and the other dilated; they vary from time to time as to irregularity and relative size. Details as to the pupillary and other ocular conditions in general paralysis may be consulted in the article of Mobèchet† and in the summary of my own‡ observations given at the close of this chapter.

Face.—The features are now usually quite flabby and of a greasy appearance, and the lower lines of facial expression partially effaced. The physiognomy, the expression of the emotions, are much altered owing to this relaxation of the lower part of the face, and to relative overaction, or even some momentary twitching spasmodic action, of the muscles about the eyebrows and forehead, which tends to produce an unwonted expression of apparently unfelt astonishment or regret. Thus, at the same moment one part of the face may seem to express one emotion, another part a different emotion.

Trunk: Limbs.—The body is often bent awkwardly forward; or it is bent to one side, sometimes this occurs with slight hemiplegia, and with or without a degree of rigidity. Atrophy of the opposite hemisphere of the brain was suggested by Baillarger§ in explanation of this symptom, when persistent.

The gait is slow, unsafe, swerving, or, later, even zigzag, with the feet kept wider apart than usual, and the steps short, groping, shuffling, or even dragging. The patient stumbles too easily over an object, and, later on, he sways and falls in attempting to turn quickly, or when starting off in rapid walk under impulse or excitement.

The power to button clothing fails; no attempt is made to write, or, the handwriting becomes illegible, or, at the very best, is extremely shaky, irregular and unfinished or even fragmentary. Often, after several attempts the patient writes a word or two, or part of a word, or several misplaced or dissociated letters thereof, and then, after a pause, makes several irregular strokes or flourishes, or puts down the pencil in momentary confusion or disgust.

* *Gazette des Hôpitaux*, May 14, 1850. 3rd series T. ii. No. 57.

† *Annales Médico-Psychologiques*, Nov. 1874, p. 325; and Jan. 1875, p. 19.

‡ See p. 54.

§ *Annales Médico-Psychologiques*, 1858, p. 168.

In this stage the reflex activity of the limbs, especially of the lower, is lessened.

Farado—contractility of the muscles of the extremities, especially of the flexors of the feet, becomes considerably and progressively lessened* according to some.

The irritability of the muscles to the galvanic current was said by Briere de Boismont† to be conserved in general paralysis with mental alienation. But, on the other hand, shortly afterwards Bucknill‡ published his view that the "excito-motory sensibility" of the limbs to the "electro-galvanic" current was diminished or lost in general paralysis. Krafft-Ebing,§ however, found that electro-muscular irritability and sensibility were retained in general paralysis, and Voisin|| declares, that, even in the second stage, the electro-muscular sensibility is intact as long as there is no softening of the cord.

In many cases the patient seizes objects, which he incessantly pulls to and pushes from himself, pulling again and again upon them if they are fixed; drawing them over him, and again thrusting them from him, if they are movable. He grasps tightly, resists passive motion, and, though very helpless, is difficult to manage. In an extreme degree, this condition is mainly found in the present stage: then hours may be spent in this monotonous and aimless handling, pulling, thrusting, rubbing of objects, and never-ceasing movements of the limbs and trunk, until the perspiration breaks forth on the heated face, the fumbling hands; yet the patient still works on with stupidly earnest gaze.

He usually becomes bedridden towards the close of this stage; or sooner, if the spinal have been early and highly marked in proportion to the other symptoms. By this time the ataxic symptoms, so prominent in the early periods, have often become overshadowed by the paretic, the muscular force is diminished, but, on occasion, considerable muscular strength can be put forth. Even those who argue that throughout "general paralysis" there is no true paralysis admit the lessening of the muscular power

* J. Lowe, "West Riding Asylum Medical Reports," vol. iii. p. 196; and Bevan Lewis, vol. v. p. 85.

† "Du diagnostic différentiel des diverses espèces de paralysie générale, à l'aide de la galvanisation localisée."—*Ann. Méd.-Psych.*, 1850, p. 603, and *Dict. des Dicts. de Médecine*, Supplément, 1851, p. 596.

‡ Jan. 1, 1852. See "A Manual of Psychological Medicine," London, 3rd edition, 1873, p. 460, where there seems to be a misunderstanding on the part of Dr. Bucknill as to Briere de Boismont's views.

§ "Allg. Zeitschrift für Psychiatrie."

|| *Op. cit.*, p. 110.

therein,* a condition sufficiently explained by the lesions of both the cellular and tubular elements of the cerebro-spinal system. At least, a paretic or paralytic element, whether general or local, now exists, or occurs, as an almost constant clinical feature.

If at last he lies in bed, the head and neck are often bent forward for hours together; or almost constantly is his head kept raised away from the pillow, the patient gazing stupidly here and there, or from time to time looking fixedly, but unintelligently, before him, the forehead at the same time being corrugated and the eyes widely open. In a few cases the lids may droop owing to temporary ptosis, supervening or not on local spasm. Often the legs are now more or less contracted and rigidly flexed, the forearms and hands flexed and lying across the chest, though the limbs perhaps are at times straightened by the patient, the speech is extremely limited, articulation indistinct, expression vacant, the tongue protruded with great difficulty, or not at all; the features often have a swollen relaxed puffy appearance, or grow thin earthy and coarser; all the natural discharges are passed involuntarily under the patient, while in many the teeth are frequently, or almost incessantly, and noisily ground together, not only by day but also making night hideous with the sound.

Motor Complications.—The epileptiform, apoplectiform, and paralytic seizures are now frequently seen, but will be described in a separate section below. As in the first stage (of the confirmed disease), so in this, the whole frame is sometimes affected with a violent muscular tremor or shivering, or the subsultus and rapid tremor may affect one side or one limb only.

Again, it is not rare to find a distinct symptomatic paralysis agitans—a true tremor cōactus—which may involve the upper extremities and head or neck, and, occasionally, the lower extremities in some degree, or may be observed on one side only, or in one thoracic limb. It may last for days or weeks. In my own experience, this has principally occurred in the second stage of the confirmed disease (third period), seldom in the first, and then only when the stages were ill-defined.

Still another morbid kinesis of this stage consists of distinct *choreiform* movements about the head and upper extremities, or more limited in their distribution. They usually last but a comparatively short time, and may readily pass unrecognized in a patient known to be restless. I have frequently noted them, and Dr. T. S. Clouston† has placed a well-marked case on record.

(β). *Sensory Symptoms.*—The optic discs become atrophied in

* J. Christian, *Ann. Méd.-Psych.*, Jan. 1879, p. 32.

† *Journal of Mental Science*, Oct. 1875, p. 421.

some cases; this following upon hyperæmia with exudation: in a few white atrophic pallor is evident, and impaired sight or blindness coexists. The ophthalmoscopic changes are specially treated of at the close of this chapter.

Fürstner * described a peculiar disorder of vision in general paralysis, for the most part in one eye only. On closing the sound eye objects placed before the affected eye are only seen confusedly; if they are objects usually coveted they are now neglected, if bright they no longer rouse attention; then, also, the writing is irregular, the lines and letters misplaced or jumbled together; the gait more affected than when the sound eye is open. Reinhardt† has published a similar case.

The other special senses may fail. Smell and taste often seem to become diminished, or lost, and cutaneous sensibility also.

Even in this stage, however, many a patient starts back with a preternatural facility and blinks when a darting movement is made towards his eyes, or he starts unduly at a sudden touch.

Cutaneous hyperæsthesia, neuralgic pains, and sensorial hallucinations are now comparatively very rare.

The palatine, faucial, and laryngeal sensibility is apparently lessened.

As a rule there is general and progressive loss of sensibility, affecting the surface of the limbs and other parts, and pinching, pricking, heat, cold, and injury, are often but little heeded. In certain cases observed by A. J. Linas (*op. cit.* p. 37), sensibility failed first in the trunk. It became more obtuse on the chest than the abdomen, on the back than the front, and disappeared elsewhere in the following order of priority;—thighs, legs, arms, forearms, neck, face, and hands. It failed earlier on the extensor than on the flexor side of the limbs, and was retained longest about the mucous orifices,—as the mouth and nose—and at the peripheral ends of the limbs.

(γ). *Eating*.—With the greater or less abolition of the sense of smell that of taste also dwindles away, and what with this, and with the intellectual failure, together with the obliteration of æsthetic feelings, of all that is self-respecting,—the eating of the patients becomes hasty, gluttonous, untidy, and disgusting. Seizing masses of food and hastily thrusting them into their mouths they endanger the integrity of their air-passages, and risk death by asphyxia, unless haply relieved by efforts of gulping.

(δ). *Cachexia*.—At the beginning of this stage the patients

* "Archiv für Psychiatrie und Nervenk," viii band, 1 heft, and ix band, 1 heft.

† Ibid.

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generally fatten, but it is a flabby fatness they put on; and afterwards they emaciate. Bedsores are very apt to form over the sacrum and trochanters, about the heels, and where the knees press together. Sometimes slowly, and in consequence of gradual failure of nutrition making itself felt in the parts which bear the weight of the body, or are exposed to pressure and perhaps to occasional excremental irritation;—at other times occurring as acute bedsores (*decubitus acutus* or *ominosus*), of either the cerebral or spinal variety.

The greasy appearance of the skin, the occasional clammy perspiration, and the noisome odour exhaled therefrom in the later periods of general paralysis, are worthy of mention. Not connected by him with general paralysis, these conditions were noticed by Sir W. C. Ellis* to forecast incurability, and the finding of excess of ventricular serum. The dull, earthy, parchment-like appearance sometimes put on by the skin has already been noticed.

Diarrhœa, furuncles, carbuncles, ecthymatous and vesicular eruptions, are frequent.

In the final periods of general paralysis, Voisin† describes the blood, under the microscope, as too thin, and the grouping of its red globules as too decided and too irregular; and viewed by the naked eye, as fluid, sticky, and as forming clots which are defective in cohesion and float in a reddish serosity. Where septicæmia had occurred he observed vibrios and bacteria in the blood, under microscopic examination.

Hæmorrhage from the mucous membranes occasionally is observed in the later periods of general paralysis.

(ε). *Othematoma*.—Occurring at any period of the disease, othematoma comes on more particularly in this and in the preceding stage.

B. MENTAL SYMPTOMS of the *Second Stage* of the *confirmed* disease (*third period* of general paralysis).

Dementia.—Much that might be said, under this heading, of the second, has necessarily been anticipated in the description of the first, stage of confirmed general paralysis. Therefore a brief summary will suffice.

In this second stage the failure of mind is very obvious, and the patients can no longer produce new ideas and new delusions. They must live upon the now disconnected or fragmentary morbid

* "A Treatise on the Nature, Causes, and Treatment of Insanity," London, 1838, p. 133.

† "Traité de la Paralytie Générale des Aliénés," 1879, p. 160.

ideas of the past. Shreds of their former delusions are now repeated almost mechanically, and, towards the close, with little or no involvement of the feelings; the loss of memory and general mental failure are extreme; exaltation is now far more rare than it was; some self-satisfaction may be observed, but more often are the patients dull stupid and unemotional; or heavy sullen morose and irascible; or querulous worried depressed and cast down. Yet, indeed, there is often rather a mere mechanical expression of these feelings; the inattention, apathy, and indifference of dementia for the most part excluding all else. Falling into a chronic state of dementia, these patients may survive for prolonged periods of time.

The mental state often passes from one phase to another; to-day repeating mechanically some fragmentary exalted delusions; to-morrow the patients are fatuous, or morose and irascible, and these delusions forgotten.

All sense of propriety or of shame is generally lost. The habits of the patients are usually filthy, their dress untidy. At last, helplessly and hopelessly demented, their rapidly failing powers of utterance are perhaps devoted to incoherent cursing and reviling, of the most foul-mouthed and obscene type. This, at least, occurs to a striking extent among soldiers, but one must bear in mind how many of that class, when in health, hold their mental acquisitions grounded upon a basis of obscenity and blasphemy.

At irregular points of the downward journey may occur congestion and heat of the head, with increased mental dulness, heaviness, and stupidity, and often insomnia; restlessness, excitement, self-smearing, furious teeth-grinding, increased ataxic disorder, and often paralytic helplessness. The mental state is often worse after the decided apoplectic or epileptic seizures described in another section.

IV. *Fourth period of general paralysis, or THIRD STAGE of the CONFIRMED disease.*

A. PHYSICAL SYMPTOMS.

The ataxic symptoms, and the paralytic, reach their greatest measure. Any movement is attended with the utmost trembling and shakiness, or, if the paralytic element predominates, the movements are feeble, ineffectual, and of small range. Seated upon a chair, the patient leans or tumbles forwards, or to one side; placed upon his feet, he stands as if rockbound, or he stumbles awkwardly, or falls with the attempt at locomotion. Therefore, perforce, he is usually bedridden. And now contrac-

tions of the limbs, particularly flexed contractions, oftentimes make their appearance and induce an unwonted deformity. These are sometimes transitory, and relax under the patient's muscular efforts, and sometimes are persistent. Now it is, especially, that bedsores, that blebs, boils, or herpetic eruptions about the extremities, appear; and now that the skin grows so dull, dirty, or greasy in appearance, despite of all ablutions.

Finally, the wretched patient is completely prostrate, bedridden, helpless, inert, of wet and dirty habits, and his cutaneous and general sensibility, as well as his coordinate motor power, are almost abolished. With special senses blunted or lost, almost or quite unable to swallow with any safety, he lies wasting, afflicted oftentimes, with diarrhœa, pulmonary lesions, bedsores, or contractions of the limbs, and still, perhaps, grinding the teeth.

If the general exhaustion from the primary disease does not carry him off, then some of its secondary, or indirect effects, such as the pulmonary, intestinal, cystic, or renal, affections—not to mention septicæmia from bedsores or other sources—will yield him a ready passport to the happy deliverance of death.

B. MENTAL SYMPTOMS.

Amentia.—The third stage of confirmed general paralysis (fourth period) is simply a profound degree of the preceding stage. The mental faculties and coherent speech-power are practically almost abolished, and the merely vegetative existence soon comes to an end. But not before we can verify the failure or loss of sensibility, both general and special, the obliteration of all moral feeling, and the retention, at the most, of a mere thin, spectral, semblance of some former phases of idea or emotion. These are but as the fragmentary remains of a downthrown dilapidated temple.

Certain Complications of General Paralysis: its temperature: and the state of the vegetative functions therein:—Not to overload the description of the several periods of general paralysis, it has been convenient to defer until the present moment the description of several important points: we refer to such complications of general paralysis as *epileptiform, apoplectiform, and paralytic seizures; meningeal hæmorrhage; aphasia; and palsy of involuntary muscles*;—we refer also to the *temperature* in general paralysis as well as to the condition therein of the *perspiration, respiration, circulation, digestion, and urinary secretion*. Finally, observations by the present writer, and by

others, upon the *pupils and eyes* in general paralysis will be appended. Several other subjects will be noticed incidentally.

Certain Complications of General Paralysis.—It is convenient to notice here special seizures to which some general paralytics are subject; from which others, however, are entirely free. They may occur at any stage of the affection, and are principally the epileptiform, and apoplectiform, to which one might add the simple paralytic.

EPILEPTIFORM SEIZURES.—The epileptiform seizures, sometimes said to occur in its advanced stages only, may in reality take place during any period of the disease, nor is it at all uncommon that the recorded case-history opens with symptoms of a convulsive nature. During the first few months, it is true, the seizures in question are comparatively rare; then after the eighth or twelfth month of the disease, if present, they are apt to become more frequent, and to continue so throughout. In some cases the attacks, absent or rare in the early part of the course, become more frequent as the disease progresses. Esquirol* long ago asserted the almost invariable attendance of convulsions upon the closing hours of general paralysis, and certainly this is frequent.

The utmost variety obtains in the degrees of their severity, extent, and duration. In a few cases a severe general convulsion occurs which is precisely like the well-developed convulsion of *Epilepsia gravior*. Clashing with the views of some, yet is this statement faithful to nature, and the characteristics sometimes described as essential points of the differential diagnosis between the convulsions of general paralysis and those of true "idiopathic" epilepsy, cannot be applied to cases such as these. The examples I have observed and noted disprove the essential, or characteristic, nature of every feature of the convulsions of general paralysis relied upon as characteristic by a living British alienist. Aug. Voisin † also observed the absolute likeness between some epileptiform convulsions of general paralysis and the seizures of true epilepsy, and found the same high and vertical ascent, the same dirotism, the same general form, of the sphygmogram, in each, immediately after the convulsion.

Several seizures, even when so severe as just described, may be repeated in close succession to one another, and the boundary of the *status epilepticus* is often crossed.

But in the majority of cases the convulsions are neither so extensive nor so severe. They often afflict the face and arm, or the face, arm, and leg of one side; and with them there may, or

* "Des Maladies Mentales," 1838, T. ii. p. 264.

† *L'Union Médicale*, 1868, p. 87.

may not, be slighter twitchings, of less extensive range, on the opposite side. When the seizure is of a still slighter nature, twitching may involve some isolated muscles, or groups of muscles, on one or both sides. These various convulsive and spasmodic attacks may occur in rapid succession, or there may be several in the twenty-four hours; or a bout, consisting thus of a variable number of seizures, may be prolonged for several hours or days, and may recur very soon, or only after the lapse of weeks or months, or not at all. In cases such as these consciousness is sometimes completely, at others partially, lost; but often remains apparently unaffected. This retention of consciousness, however, is far more apt to obtain in those instances where the convulsions are resolved into local convulsive twitching of several groups of muscles, or of one group; convulsive twitching which may continue for hours or days without ceasing—may transfer itself to other parts—or may alternate with convulsive twitches elsewhere—and which constitutes one of the most clearly defined complications of general paralysis.

Then, again, it is a highly characteristic condition when the patient is seized with convulsions, at first tonic, then clonic, affecting the face mainly on one side, although the other side may also be affected in less degree;—when, with this, the muscles of the upper extremity take on marked convulsive action, which perhaps also slightly invades the lower extremity, and continues for several minutes without, or with only slight or moderate, loss of consciousness;—when, after this, there is a calm, broken only by spasmodic jerking about the mouth or the eyes, together with twitching of the forefinger and thumb, or by jerking at the shoulder joint;—when, next, perhaps, a recurrence of the former widely-spread unilateral convulsion takes place; or a short, sharp tonic spasm, drawing the head violently to one side, distorting the features, and stiffening the upper limb in various directions, is followed by rapid clonic successions;—when, possibly, the spasmodic jerkings now temporarily emigrate to new fields, invading fresh groups of muscles, perhaps on the opposite side of the body, and, for the time, abandoning those they formerly occupied; and, when, after a repetition of convulsions such as these, especially of the more strictly localized ones, there is more or less paralysis of the face and upper limb on the side principally convulsed, and, perhaps, in slight degree, of the lower limb also, as well as considerably heightened axillary temperature, which, of the two, is higher on that side which was mainly convulsed at the first and then, temporarily, paralyzed. Conjugated deviation of the head and eyes is often observed here; they

usually turn *from* the paralyzed side. During and between the actual seizures certain muscles of the limbs, neck, or trunk may be rigidly contracted and unyielding. Thus may the forearm be rigidly flexed across the chest, the fingers and thumb buried in the palm, the head and neck distorted.

Almost invariably the above paralysis clears up in a few hours or days. While paralyzed, the members, usually flaccid, are occasionally rigid. But it is not only after the most severe and extensive convulsions that an *Epileptic paralysis* is seen; often a very partial and a very incomplete paralysis, essentially of the same nature as so-called epileptic hemiplegia, obviously follows the more intense of the localized and limited spasms, —spasms limited, for example, in distribution to the face, or the face and tongue, or an upper limb, or to parts of either of these. In truth, had we sufficiently delicate means of gauging it, some minute local loss of motor power would no doubt be found in the part affected after every spasm, however slight. For paralysis follows these convulsions and spasms as the shadow follows the body.

The temperature rises during and immediately after, sometimes also immediately before, these convulsive attacks; the elevation of temperature continues for some hours and may be very considerable after severe fits.* During the attack the pulse increases in frequency, and is often irregular; during it, also, perspiration and discharge of urine and fæces are common occurrences. Huppert† frequently found albuminuria after epileptiform attacks in general paralysis.

Yet often the attacks are only very slight, and of a nature analogous to the *petit mal* of idiopathic epilepsy. Thus they may merely consist of a sudden pallor with mental confusion; or of dilatation of the pupils, with drawing of the head to one side or of the mouth agape; or of sudden fixation of the lineaments or an expression as of shock, together with cold perspiration, or with the muttered automatic repetition of coherent or of incoherent phrases. No doubt these are often mistaken for syncopal attacks.

Mr. Newcombe‡ avers that the ages at which general paralysis supervenes in no way influences the liability to epileptiform seizures; but that, on the average, the younger the patient attacked by general paralysis the later on in its course do these seizures first appear.

* See also the section on the temperature in general paralysis.

† *Journal of Mental Science*, Jan. 1878, p. 602 (Abstract).

‡ West Riding Asylum Reports, vol. v. p. 197.

After severe seizures two conditions are usually noticed; (*a*) the mental state has further deteriorated; and (*b*) the general motor disorder and failure have become more marked. These are not obvious, however, after the slighter attacks.

APOPLECTIFORM SEIZURES.—Under this heading are often included cases in which apoplectiform symptoms are associated with convulsions, and strictly speaking this is correct. But it is convenient to consider separately the apoplectiform seizures occurring without convulsions. Reference is not made here so much to the apoplectiform seizures occasionally observed at the very outset of general paralysis, or upon the heels of which it seems to swiftly follow, as to those which often chequer its later morbid career. Well-marked apoplectiform seizures without convulsions are less frequently observed in general paralysis, than the epileptiform, from many of which they are mainly distinguished by the absence of convulsive symptoms. These apoplectiform attacks may come suddenly and crushingly upon the patient; on the other hand they are often preceded by the occurrence or increase of insomnia, restlessness, or excitement, by redness or turgidity and heat of face and head, or by a mental heaviness dulness and stupidity, and an aggravation of the ataxic and parietic disorders, as shown in the locomotion and in all the other movements. They are often recurrent, vary from the slightest shade to the most extreme degree of apoplectiform unconsciousness and coma, are immediately preceded, and are accompanied by some elevation of temperature, are partially expressed in turgid congestion of the face, heated head and skin, a rapidly heightened axillary temperature, sometimes dilated pupils, and involuntary passage of urine or of fæces; and are not infrequently followed by temporary paralyses, of which the most common is a partial, or, again, an incomplete hemiplegia, and with this, conjugated rotation of the head and eyes to the opposite side may be found, while an eschar may, or may not, appear on the buttock of the palsied side.* In some cases all the limbs appear to be powerless.

Mental dulness, heaviness, drowsiness, or restlessness generally succeed the decided attacks, and hours, or days, or even weeks, but usually several days, elapse before the patients return to their former state; and yet the return is scarcely to their former state, but rather to a lower level on the incline of dissolution, and after each such attack the mental and motor symptoms, as a rule, are slightly worse.

* See cases at end of this work.—Also V. Hanot, "*Comptes Rendus des Séances et Mémoires lus à la Société de Biologie*," 1872, p. 61, &c.

Paralytic Seizures—Meningeal Hæmorrhage—Aphasia. 41

In fatal seizures associated with unilateral palsy, meningeal congestion or hæmorrhage, cerebral congestion, or a predominance of the ordinary histological changes of general paralysis, are usually found affecting the brain and meninges on the side opposite to the paralysis.

SIMPLE PARALYTIC SEIZURES.—The simple paralytic seizures, comparatively rare in their marked degrees, are those in which sudden motor collapse and decided local paralysis, ay, even at times a hemiplegia, occur without convulsion or spasm of any kind, as far as is known, and without any observed indications of coma. But they are usually accompanied at first by some pallor, or facial expression as of shock, or even by a momentary mental confusion and slight obnubilation, making one think of an epileptoid basis. Almost invariably do these paralysees clear up and vanish rapidly, or, at least, without any very prolonged delay.

Over and above the more or less generalized paresis from which the patients may suffer, there is sometimes found a very slight and very transient local loss of power; discoverable only by searching examination; independent, as far as known, of any epileptiform, or apoplecticform seizures; and possibly affecting now this part and now that, as, for example, one, or several muscles about the face, tongue, hand, or upper limb.

These transient paralytic seizures are widely distinct from those somewhat persistent hemiplegias occasionally observed, and already mentioned in a preceding section.

MENINGEAL HÆMORRHAGE.—Meningeal hæmorrhages, arachnoidean, or sub-arachnoidean, are not rare in general paralysis. They give rise sometimes to no special symptoms; sometimes to apoplectic, or epileptiform symptoms and paralysees; sometimes they terminate fatally in a few hours or days; while their more usual ending (if large) is the formation of "arachnoid cysts."

In one case under my care where extremely large and thick double arachnoid cysts were found after death, the patient, from an early period, was more or less restless, sleepless, excited, noisy, destructive, and at times boisterous, filthy, mulishly obstinate, resistant to every form of necessary care and management, self-helpless and neglectful, utterly incoherent, and, indeed, rarely uttering aught save unintelligible sounds emitted in a noisy monotone. Severe convulsive seizures befell from time to time, and sub-intrant attacks of the same somewhat abruptly terminated life.

APHASIA.—Occasionally a general paralytic is smitten with aphasia, and this is usually sequential to a seizure of one of the

42 *Temperature of Body in Paralysis of the Insane.*

three kinds above described. See cases by Dr. Clouston,* and by the present writer.†

Symmetrical lesions of the posterior and inferior parts of the third frontal convolutions on both sides, and a slight lesion on the anterior and inferior part of the left one were observed in one case.‡ The lesions were old, and consisted of localized softening and discoloration, almost limited to the grey cortical substance.

But no strictly limited lesions of this kind are observed in some of the cases aphasiac, in general paralysis.

True aphasia, not the terminal speechlessness of general paralysis, is referred to here.

Involuntary Muscles.—Loss of power may occur in the involuntary muscles, as in those of the bladder or intestine, producing respectively ischuria vera and constipation; and for the relief of which strychnia is recommended by Verga (Abst., *Journ. Ment. Sci.*, April, 1875, p. 139).

THE TEMPERATURE IN GENERAL PARALYSIS.—Strictly speaking, this forms part of the description of the stages of general paralysis, but it was thought well to defer it until now so as (1) to allow of a prior description of the special seizures in general paralysis, in which the temperature-variations play so important a part; and so as (2) to take a general view of the subject.

In the maniacal attacks of the early stage L. Meyer found the temperature increased; and markedly so at the vertex of the skull.

Clouston§ observed that of the several forms of insanity the *mean* temperature, of the group of patients representing each, was highest in general paralysis;—that in it, also, the average *evening* temperature was always *above* the average morning temperature, the difference between the morning and evening temperature being greater in it than in any other of the several forms of mental disease; and that “the temperature is high in the first stage of general paralysis, lower in the second stage, and again very high in the third. The evening temperature is most increased as compared with the morning temperature in the third stage, and least in the second.”

In general paralysis I have *almost* invariably found the *average evening temperature* in excess of the average morning tempera-

* T. S. Clouston, *Journal of Mental Science*, Oct. 1875, p. 421.

† W. Julius Mickle, *Journal of Mental Science*, Jan. 1876, pp. 569 and 583.

‡ Billod, *Annales Médico-Psychologiques*, May, 1877, p. 339. A case by Cullerre, *Ibid.* 1878.

§ *Journal of Mental Science*, April, 1868.

ture, but this is not an absolute rule throughout the whole duration of every case, as may be seen by glancing at the table at page 37 of my contribution on the subject in the *Journal of Mental Science* for April, 1872. In the rare cases in which the *average* morning temperature exceeds the *average* evening temperature, its excess is but slight, lasts only a comparatively short time, and is restricted to the earlier periods of the disease. In each instance the averages in question are the averages of the axillary temperatures on thirty successive mornings and evenings, taken with every pains and precaution, and often for many months continuously so as to obtain a number of averages at different periods.

The following were the general conclusions at which I* arrived:—

That in the middle and later stages of general paralysis of the insane:—

1. A rise in the temperature often accompanies a maniacal paroxysm.

2. A rise in temperature often precedes and announces the approaching congestive or convulsive seizures, and nearly always accompanies them.

3. When these states are prolonged (congestive, or maniacal), the associated elevation of temperature is usually prolonged also.

4. Defervescence of temperature, after its rise with excitement or with apoplectiform attacks, often precedes the *other* indications of toning down to the usual state.

5. Moderate apoplectiform attacks, or moderate maniacal exacerbations, are, however, not invariably associated with increased heat of body.

6. A transitory rise in temperature may occur without any *apparent* change in mental or physical state to account for it.

7. The evening temperature is usually higher than the morning temperature in general paralysis, and an absolutely high evening temperature occurs in cases rapidly progressing towards death.

8. A *relatively* high evening temperature seems to be of evil omen, even when *not absolutely* very high.

9. Rapidly progressing cases may show temperatures above the average both in the morning and evening, for a long time before any complication exists.

10. Gradual exhaustion may pass on to death, in general paralysis, with an average morning temperature normal, or nearly so, throughout, except when raised temporarily by the special attacks to which general paralytics are subject.

* *Journal of Mental Science*, April, 1872, pp. 45, 46.

44 *Temperature in Apoplectiform Seizures.*

11. The onset, especially, of pulmonary complications, or of hectic from bedsores, is marked by much heat, and when death is accelerated by the former, the temperature and pulse run high, often, however, sinking somewhat before death, whilst respiration then becomes very rapid.

These conclusions as to the state of the temperature in the apoplectiform and epileptiform attacks of general paralysis have been confirmed by several observers, and among other recently by Dr. Riva.* Dr. Saunders† published a case some years ago.

Dr. D. Hack Tuke‡ subsequently published certain thermometrical observations made by Dr. Wm. Macleod, which in some respects were confirmatory of the conclusions above cited.

It is particularly worthy of note that the axillary temperature during and immediately after an apoplectiform attack is higher on the side of the body which alone, or principally, is paralyzed (if there be paralysis). And that after an epileptiform seizure of a more or less unilateral character the axillary temperature is higher on the side in which convulsion mainly appears, and in which some paresis or paralysis reveals itself immediately thereafter. I base this general rule upon a very large number of personal observations.

The Temperature in Apoplectiform Seizures.—One or two examples will suffice.

In a fatal apoplectiform seizure in the course of general paralysis, the following is an illustration of the course taken by the temperature. On the first day, with extreme apoplectiform cerebral congestion, left hemiplegia, also, came on. The patient rallied slightly, but the congestive symptoms returned in full force, and the pulmonary congestion began to pass over into hypostatic pneumonia.

| | Temperature in left axilla. | Temperature in right axilla. |
|---------|--------------------------------|---------------------------------|
| 1st day | 103·2° Fah. | 103·1° Fah. |
| 2nd „ | 103·2° | 102·9° |
| 3rd „ | 101·8° | 101·7° |
| 4th „ | 100·2° | 100·3° |
| 5th „ | 102·5° | 101·3° |

* “*Rivista Sperimentale*”; quoted *Journ. Men. Sci.*, April, 1879, p. 125. See also *Journal of Mental Science*, Oct. 1875, p. 424;—case by Clouston. Société de Biologie, 1873, V. Hanot.—Acad. de Médecine de Paris; Meeting of May 16, 1877, and *Ann. Méd.-Psych.*, July, 1877, p. 131;—case by Magnan. Voisin, “*Traité de la Paralyse Générale des Aliénés*,” 1879, pp. 211 and 218.

† Quoted by Maudsley, “*Pathology of Mind*,” 1879. Third Edition.

‡ “*Manual of Psychological Medicine*,” 1873, p. 325.

The difference between the temperatures of the two axillæ lasts sometimes for two or three days in the milder and non-fatal apoplectiform attacks. In these, however, the rise in temperature is not so extreme. Thus, in one case, after an attack of this kind with left hemiplegia, the temperature in the left axilla was 101.2° , in the right, 100.2° .

As an exception to the general rule, that, during and just after an apoplectiform seizure with paralysis the temperature is higher on the side thus temporarily paralyzed, a case may be mentioned in which, with left hemiplegia of this nature, the thermometer rose in the left axilla to 99.4° , and in the right to 100° .

Many examples of the behaviour of the temperature preceding, during, and just after, apoplectiform attacks in general paralysis are reported by myself in the paper already mentioned (p. 38, *et seq. Journ. Mental Science*, Apr. 1872), to which I would crave the attention of the reader.

The Temperature in Epileptiform Seizures.—After convulsive seizures in general paralysis, especially those that leave decided traces of palsy in the parts most convulsed; (1) the general temperature as a rule is increased, and (2) is higher on the side affected when the convulsions have been unilateral; and the temperature may remain higher for a day or two on that side than on the other.

But as to the former of these points—the general increment of body heat—it must be added that when the patient falls day after day into epileptiform convulsions the temperature at last is found in certain cases, to stand below the normal, thus forming exceptions to the general rule.

And as to the second point also,—namely the higher relative elevation of the temperature on the side convulsed—it must be conceded that the temperature, after being higher on the side most convulsed, *may*, a day or two afterwards, sink to a lower thermometric level on that side than on the other, and this may even occur before all convulsions have ceased. Thus, in one general paralytic in whom sub-instant convulsions produced temporary right hemiplegia with a right axillary temperature of 99.4° , and a left axillary temperature of 99° , the thermometer had sunk three days later to 97.8° in the right axilla, and to 98.3° in the left, although one or two mild convulsions still occurred each day. Although the right hemiplegia still persisted the temperature of the right side, at first the higher, had now fallen to a point slightly below that of the left.

Again, in another case, six epileptiform seizures were followed by left hemiplegia, the thermometer reading 102.5° in the left,

and 102° in the right axilla, or half a degree higher in the left; but three days later the left temperature, though still abnormally high, was one-fifth of a degree lower than the right, that of the left axilla being $99^{\circ}6'$, that of the right $99^{\circ}8'$,—the left hemiplegia being still highly marked, but no convulsion having occurred for nearly twenty-four hours.

In these cases hemiplegia, as usual, befell the same side as unilateral convulsions, or as convulsions predominant on that side.

As for the simple *paralytic seizures* unconnected, as far as known, with marked apoplectiform or epileptiform attacks;—In one example, a few hours after the onset of a left hemiplegia of this kind a thermometer marked 99° in the left, and $97^{\circ}8'$ in the right axilla (difference $1^{\circ}2'$). Two days later, the palsy rapidly clearing up, the left axillary temperature was $97^{\circ}9'$, the right $97^{\circ}2'$ (difference, $7'$).

Some hours after a similar, but slight, seizure of sinistral hemiplegia the thermometer stood at $97^{\circ}8'$, in the left axilla, at $97^{\circ}3'$ in the right (difference $5'$).

Another patient suffered from an attack of dextral hemiplegia of this nature on the 9th, and the right temperature was $98^{\circ}9'$, the left $98^{\circ}7'$. The paralysis persisted more or less for some time, and on the 11th the temperatures were; right axilla $99^{\circ}3'$; left, $99^{\circ}1'$; on the 13th, right, $98^{\circ}8'$; left, $98^{\circ}6'$; and by the 16th, right, $98^{\circ}3'$; left, $97^{\circ}4'$. But frequent slight convulsive quiverings now began to play about the right side, and especially in the face, and by the 21st the right temperature was $97^{\circ}7'$; the left $96^{\circ}7'$; and by the 25th the right was 98° ; the left, 97° ; the next phase being that dextral spasm and paralysis were augmented on the 27th, and the temperature had risen to $99^{\circ}6'$ in the right axilla, to $99^{\circ}2'$ in the left.

It may be added that in a case of sudden right hemiplegia from embolism of the left middle cerebral artery, in a general paralytic, I found the temperature between one and two hours afterwards to be $101^{\circ}5'$ in the right, and $101^{\circ}3'$ in the left axilla.

The Thermometrical Fluctuations and Pyrexia in General Paralysis.—Having thus fully entered into the subject of the average temperatures throughout the course of general paralysis, and into that of the thermometrical perturbations attending its apoplectiform, epileptiform, and other complications, we may briefly refer to irregular fluctuations and pyrexial movements independent of special seizures.

If we examine the temperature charts of general paralytics for elevations above the normal temperature, we find that they occur

irregularly. From the following numbers, however, a slight deduction must be made, inasmuch as the readings at, as well as those above, 98.4° are included.

For example, in one general paralytic with considerable and protracted maniacal excitement in the second stage of the confirmed disease:—

During one period (*a*) the morning temperature on fourteen of forty-six mornings was at, or above, 98.4° : then (*b*) during the next three months a rise above this line was observed only on the 1st and 2nd day of one month, on the 25th and 26th of the next, and on the 6th, 8th, and 9th of the next.

The evening temperature, taken only during the former (*a*) of the above two periods, was at or above the same line (98.4°) on three-eighths of the evenings, and the rise occurred at irregular intervals, and on from one to three successive nights on each occasion.

Another, in the second (and early third) stage, had at one period no morning temperature above 98.4° , whereas on almost half the evenings the temperature was above that level.

Three months later, the morning temperature occasionally rose to the line (98.4°).

Eight months later, when the patient was much more demented and helpless, and had suffered *interim* from severe epileptiform convulsions, three-fourths of the morning, and all the evening, temperatures were above the line.

Another, in the final stage, whose morning temperature was taken regularly for four months before his death, had a temperature above the line (98.4°) on one-fourth of the mornings.

His evening temperature, taken about four and three months before death, was above the line twenty-three times out of thirty-nine, remaining above it for from one to four successive nights at irregular intervals.

A somewhat rapidly advancing case of general paralysis of the hypochondriacal form, during one period had nearly all the morning temperatures above the line, and *all* the evening temperatures either above, or at, it.

Three months later, both morning and evening temperatures, generally above, were occasionally at or below the line.

Eight months later, the same was still true of the morning temperatures, but *all* the evening temperatures were now above the line.

In another patient, one who was in the second stage (third period of this work) of classical general paralysis, both the morning and evening temperatures were above the line in two-thirds of

48 *General Paralysis of the Insane—Unilateral Sweating.*

the observations, remaining so during from one to four mornings or evenings in succession, at irregular but closely set intervals.

With reference to the pathological import of the greater tendency to elevation of vespertine than of matutine temperature in these cases, it must be borne in mind that, on the contrary, in health the morning temperature usually exceeds the evening.

The above morning and evening temperatures were for the most part taken only about 10 or 11 A.M., and 8 P.M., respectively.

In certain cases the temperature becomes quite subnormal, and even low (90° — 95°) for a day or two, or longer, before death.

SWEATING in General Paralysis.—The general state of the skin and of its secretions has already been mentioned when treating of the third and fourth periods (second and third stages) of the affection.

In the *Journal of Mental Science* for July, 1877, p. 196, I have described three cases of general paralysis in which there was the phenomenon of unilateral copious sweating of the face and head (*hyperidrosis unilaterialis et partialis*). The unilateral perspiration of the face occurred under somewhat dissimilar conditions in these three cases of general paralysis: in one, without any local paralysis or convulsion, but associated with old-standing disorganization of the eyeball of the same side; in another, the unilateral perversion of secretion accompanied unilateral convulsions and hemiplegia of the corresponding side; while in the third it occurred some time after a peculiar change in the mental state, and at first with very slight and very transitory unilateral facial paralysis.*

In the third section (p. 206) of that article I treated at some length of the probable pathology of this condition of local hyperidrosis in general paralysis. Views similar to those theoretically suggested as to the existence of sudoriparous secretory nerves and nerve-centres, have recently been apparently established by the experiments of Luchsinger, Nawrocki, Adamkiewicz and Vulpian. As stated in my article (p. 212):—"Whilst, therefore, both the sympathetic and the cerebro-spinal system influence secretion, and both the vaso-motor and the so-called secretory nerves, it is mainly to the cerebro-spinal centres that we may perhaps look for the nervous influence on secretion, and mainly to the secretory nerves for the more immediate channel of that influence. It is only necessary to recall the cerebro-spinal morbid changes in general paralysis, to find the origin of a reflex morbid influence upon secretion through the cerebro-spinal fibres

* Second and third cases transposed in summary at p. 196, *loc. cit.*

supplied to the glandular system." The sweat-secretory nerves of the face are perhaps derived from sympathetic fibres which either come from the superior cervical ganglion by the route of the vertebral artery or from the bulbar and pont-Varolian regions.

In two cases of general paralysis Dr. F. Servaes * observed bloody sweating, limited to the head and face.

CIRCULATION.—In the early stages of some cases of general paralysis the pulse is full and hard, the beat of the heart powerful, the first sound clear and full, the second accentuated, the arterial tension increased. This condition, however, is very far from being always present, or persistent. The pulse, indeed, has in many cases an increased frequency, a softness and unresisting feel, which, together with the characters of the heart-sounds, tell of diminished arterial tension. Finally, in some cases, or at some periods, both the pulse and heart-sounds may possess normal characters.

Even in the early period, Voisin† finds indications of vaso motor paralysis in the full compressible pulse, whose sphygmogram displays some elevation of the percussion-stroke, a plateau at the summit, oscillations in the line of descent, and a variable degree of dicrotism. This condition increases in proportion as the malady progresses. In the final periods the tracing is reduced to a slightly, and irregularly, wavy line.

On the other hand, a sphygmographic tracing similar to that found immediately after cold immersion, was thought by Dr. George Thompson,‡ to be characteristic of general paralysis, and indicative of persistent spasm of the arteries and capillaries in that disease. The absolute validity of this inference was denied§ at the time, and cannot be acceded to now. Dr. G. H. Savage|| also has found great variety in the pulse-tracings in general paralysis, and none characteristic.

As to the pulse-frequency in general paralysis, and the comparative relations between the morning and evening pulse-rates, a mass of observations extending over many months, and incidentally summarized under the headings of *average morning* and *average evening pulse*, are contained in my¶ previously mentioned contribution. There, also, are linked together

* "Allgemeine Zeitschrift für Psychiatrie," 1863; and Abstract, *Journ. Ment. Sci.*, 1864, p. 393.

† *Op. cit.*, pp. 62, 118, 153.

‡ West Riding Asylum Medical Reports, vol. i. p. 58. The same view, since, in *Journ. Ment. Sci.*, Jan. 1875, p. 581.

§ *British and Foreign Medico-Chirurgical Review*, Jan. 1872, p. 33.

|| *Journal of Mental Science*, Apr. 1875, p. 149.

¶ *Ibid.* April, 1872, p. 31.

simultaneous observations on the temperature, pulse, and respiration in a number of cases, and at various parts of the course, of general paralysis.

From these it may be seen that in general paralysis there is a tendency to an increase of the pulse-frequency throughout the whole nycthemeron. From these, also, it may be seen that the *average* pulse of the evening is almost invariably accelerated to a greater speed than that of the morning. And this notwithstanding that the patients had retired to rest before the evening pulse was taken, whereas in the morning they were usually up and about, and only assumed the recumbent posture for a few minutes, so as to facilitate the simultaneous observation of the axillary temperature.

The average evening pulse exceeded the average morning pulse in every case, except during two portions of the course of one single case. The evening excess varied from one to thirteen beats per minute, and the mean average of all the evening excess-rates was about five and a half;—or, including the exceptional case, the total mean average evening excess in pulsations was about four and two-thirds per minute.

| Average Morning Pulse. | Average Evening Pulse. | Evening excess, or diminution. |
|---------------------------|---------------------------|-----------------------------------|
| 68 | 69 | + 1 |
| 72·9 | 84 | + 11·1 |
| 73·2 | 84 | + 10·8 |
| 76·9 | 89·4 | + 12·5 |
| 94·9 | 100·3 | + 5·4 |
| 72 | 76·3 | + 4·3 |
| 73 | 83 | + 10 |
| 84 | 87·2 | + 3·2 |
| 80 | 84·3 | + 4·3 |
| 85·4 | 86·6 | + 1·2 |
| 111 | 115 | + 4 |
| 93 | 95 | + 2 |
| 108 | 121 | + 13 |
| 72·7 | 76·3 | + 3·6 |
| 71·8 | 75·4 | + 3·6 |
| 120 | 124 | + 4 |
| 84·2 | 84·3 | + 1·1 |
| 88·7 | 82·6 | — 6·1 |
| 82·4 | 79 | — 3·4 |

RESPIRATION.—My observations of the average respiration frequency in general paralysis have already been incidentally referred to.

In general terms;—the respiration is normal in this disease

VOYAGE

except during and immediately after the special seizures already described; or, in connection with pulmonary and other visceral lesions, directly or indirectly dependent upon the primary disease; or, again, in association with accidental complications.

In one case recently published by myself* there was decided "respiration of ascending and descending rhythm" (Cheyne-Stokes' respiration).

Here "the peculiar respiration was subsequent to the first appearance of hypostatic inflammation of the lungs; and, moreover, occurred while the patient was prostrated by epileptiform convulsions, and was being supported by nutritive enemata; departed before the cessation of the convulsions, or of this mode of feeding; and did not return, or at least was not observed, during the remaining ten weeks of life." The aorta was healthy, and the heart nearly so.

In a second case of general paralysis I† observed a modified Cheyne-Stokes' respiration two days before death. The patient was then partially comatose, and had dextral hemiplegia, following upon right unilateral convulsions.

In a third general paralytic, a similar modification of the respiratory rhythm appeared on the day of death; associated with it were coma, low axillary temperature, general muscular relaxation and flaccidity. Besides the brain disease, phthisis pulmonalis and tubercular spinal meningitis, were present in this case. A fourth case came under my notice.

Posterior pulmonary congestion and œdema, lobular and hypostatic pneumonia are very frequent towards the close of life, and now and then localized pulmonary gangrene occurs, either after the inhalation of food, or following gangrenous embolism due to absorption from the site of foul bedsores, and transportation into the lungs, of thrombi impregnated with the ichor; as, indeed, was first observed by Foville.‡

The coincidence of visceral gangrene and external gangrenous affections was exemplified in a case under Dr. J. M. Charcot,§ and reported by Dr. Benjamin Ball, in which gangrenous erysipelas was accompanied by gangrenous dissecting pneumonia, a sequestrum of pulmonary tissue being surrounded by a gangren-

* *British Medical Journal*, Aug. 31, 1878, p. 308.

† *Ibid.* footnote, p. 312.

‡ "Dict. de Méd. et Chir. Prat.," T. i.

§ *L'Union Médicale*, N.S., T. v. Jan. 26, 1860, p. 162, and Jan. 28, 1860, p. 182. Charcot and Ball also report cases of similar import by;—Verneuil and Houël; Ph. Boyer; Rayer; Fournier; Carswell; Lebert; Virchow.

ous zone. Their suggestion that the pulmonary gangrene depends on the entry either of septic fluid, or of blood-clots charged with gangrenous sanies (ichorous embolism), into the blood stream from the site of external sphacelus, was anticipated by Foville, as we have already seen.

I am convinced that in some cases localized pulmonary gangrene in general paralysis follows the inhalation of food into the smaller bronchial passages and pulmonary vesicles.

DIGESTION and Appetite for Food.—The alimentary functions are usually active and well-sustained throughout the greater part of the course of general paralysis, and as a rule the appetite for food remains good until the last. Often the patients eat eagerly and gluttonously, and stuff themselves if permitted an unlimited allowance;—removed from the immediate presence of food, they show no morbid desire for gormandizing, and in certain mental phases they may even refuse to eat. Their scruples, usually overcome by persuasion, occasionally yield only to the enforcement of alimentionation by mechanical means.

Dr. J. Workman * deems an increased keenness of appetite as highly significant in a diagnostic point of view.

In the later stages diarrhoea is often troublesome and at times intractable. The stools then are thin, brown or yellowish, and contain mucus and undigested particles of food. Local muco-enteritis, mucous congestion, or ileo-colonic ulceration are usually the associated pathologico-anatomical conditions.

Constipation and quasi-paralytic conditions of the intestines in general paralysis have been referred to in a previous section.

URINARY EXCRETION.—In 1845 Dr. Bence Jones indicated a great deficiency of phosphates in the urine of general paralytics.

The same diminution of phosphates in the third stage of general paralysis was one of the outcomes of some investigations published by the late Dr. Alex. J. Sutherland,† the analyses having been made by Dr. L. Beale. A minus quantity of albumen was found in the blood, simultaneously.

Speaking of the urinary excretion in general paralysis, Mr. A. Addison‡ said that “in states of excitement the quantities of chloride of sodium, urea, phosphoric and sulphuric acids are less than in the quiescent state,” and less, also, than the quantities excreted in the same time in a state of health. “In the demented cases (the) quantities are about normal—some slightly above—and some below the mean” (pp. 444 and 449).

* *The Canada Lancet*, Sept. 1878, p. 3.

† “*Medico-Chirurgical Transactions*,” London, 1855, p. 261.

‡ *British and Foreign Medico-Chirurgical Review*, April, 1865, p. 425

According to Griesinger,* Sander found the excretion of urea to be small in general paralysis.

It was found by Dr. J. Merson,† that in the urine of general paralytics the quantity of urea was usually much increased; the quantities of chlorides and of phosphoric acid notably diminished; and the amount of sulphuric acid about normal. Also, that the *mean* specific gravity did not materially differ from that of health, and that the *absolute* quantity of urine, though slightly below that of health, was, in truth, slightly in excess of the latter, if estimated according to body-weight.

Dr. S. Rabow‡ states that "general paralytics usually secrete, in the so-called first stage, an increased quantity of urea, and, taking into account the greater consumption of food, more urea and chlorides than healthy individuals. With advancing dementia, the quantity of urine diminishes, as well as the absolute amount of urea and chlorides, whilst the specific gravity appears to be increased, and a turbidity, due to urates, seldom fails to be present. When removed by a catheter, though at first acid, it rapidly becomes alkaline."

Rabenau§ finds albuminuria more common in general paralysis than in any other cerebral disease, and believes it to be independent of any change in the kidneys. On the other hand, Rabow|| agrees with Richter¶ in opposing the view of Rabenau and in concluding that albumen is not a frequent pathological constituent of the urine in general paralysis, and that if it *is*, "it is not connected with the cerebral disease."

Max Huppert,** however, found albuminuria after the epileptiform seizures in general paralysis.

Seeing the frequency of renal disease in general paralysis, one would expect to find albuminuria more often than is the case.

PUPILS AND EYES.—In the description of the several stages the condition of pupils and retina has been briefly referred to, but it was convenient to defer treating the subject in detail. And here may be summarized the general results of an investigation into the pupillary condition in general paralysis made by myself in 1872. A considerable number of cases were watched for a

* "Mental Pathology and Therapeutics." Trans. 1867, N.S.S.

† West Riding Asylum Reports, vol. iv. p. 63.

‡ "Archiv für Psychiatrie und Nervenkrankheiten," Band vii. 3 Heft, p. 62, 1877; Trans. *Journal of Mental Science*, July, 1877.

§ "Archiv für Psychiatrie," &c., 1877. Abstract, *Journ. Ment. Sci.*, Jan. 1878, p. 602.

|| *Op. cit.*

¶ "Archiv für Psychiatrie," &c., Band vi. 2 Heft.

** Ibid. Band vi. vii. 1 Heft.

long period, and the state of the pupils examined and noted almost every day, the general mental and motor condition being also noted simultaneously.

(a.) Where the patients are at one time excessively elated, and at another extremely depressed, the condition of the pupils was alike, *as a rule*, under the two mental conditions. In several such cases they were dilated, very sluggish, but not differing very much in size.

(β.) In the period succeeding that of acute excitement, or into which patients drift from early expansive delirium, namely, the period corresponding to the second stage of *confirmed* general paralysis, I found the following conditions of the pupils:—Under each heading the several pupillary modifications are mentioned in their *order of relative frequency*. These modifications are described under the headings of *symmetry, size, mobility, and shape*.

| | | | | | | |
|--|---------------------|---|---|---------------------|--|-----------------------------|
| Symmetry | . | Pupils equal, but one or the other <i>occasionally</i> the larger. Left pupil the larger. | | | | |
| Size | . | <table border="0"> <tr> <td>Pupils rather small.</td> <td rowspan="3">} in equal numbers.</td> </tr> <tr> <td>" of natural size.</td> </tr> <tr> <td>" much dilated.</td> </tr> </table> | Pupils rather small. | } in equal numbers. | " of natural size. | " much dilated. |
| Pupils rather small. | } in equal numbers. | | | | | |
| " of natural size. | | | | | | |
| " much dilated. | | | | | | |
| Mobility | . | <table border="0"> <tr> <td>Pupils very sluggish, or almost immobile, persistently.</td> <td rowspan="3">} in equal numbers.</td> </tr> <tr> <td>Pupillary sluggishness varying much in degree from time to time.</td> </tr> <tr> <td>Pupils moderately sluggish.</td> </tr> </table> | Pupils very sluggish, or almost immobile, persistently. | } in equal numbers. | Pupillary sluggishness varying much in degree from time to time. | Pupils moderately sluggish. |
| Pupils very sluggish, or almost immobile, persistently. | } in equal numbers. | | | | | |
| Pupillary sluggishness varying much in degree from time to time. | | | | | | |
| Pupils moderately sluggish. | | | | | | |
| Shape | . | <table border="0"> <tr> <td>Pupils,—both irregular.</td> <td rowspan="2">} in equal numbers.</td> </tr> <tr> <td>" mutably irregular.</td> </tr> </table> | Pupils,—both irregular. | } in equal numbers. | " mutably irregular. | |
| Pupils,—both irregular. | } in equal numbers. | | | | | |
| " mutably irregular. | | | | | | |

(γ.) Where the condition described under (β) was merging into a more complete dementia, the following were the pupillary modifications:—

| | | | | | | | |
|-------------------------------------|---------------------|---|-------------------------------------|---------------------|--------------------------|------------|------------------------|
| Symmetry | . | Pupils equal, but <i>either one</i> at times somewhat or slightly the larger. | | | | | |
| Size | . | <table border="0"> <tr> <td>" equal, or right pupil the larger.</td> <td rowspan="4">} in equal numbers.</td> </tr> <tr> <td>" of about natural size.</td> </tr> <tr> <td>" dilated.</td> </tr> <tr> <td>" slightly contracted.</td> </tr> </table> | " equal, or right pupil the larger. | } in equal numbers. | " of about natural size. | " dilated. | " slightly contracted. |
| " equal, or right pupil the larger. | } in equal numbers. | | | | | | |
| " of about natural size. | | | | | | | |
| " dilated. | | | | | | | |
| " slightly contracted. | | | | | | | |
| Mobility | . | <table border="0"> <tr> <td>" sluggish.</td> <td rowspan="2">} in equal numbers.</td> </tr> <tr> <td>" somewhat sluggish.</td> </tr> </table> | " sluggish. | } in equal numbers. | " somewhat sluggish. | | |
| " sluggish. | } in equal numbers. | | | | | | |
| " somewhat sluggish. | | | | | | | |
| Shape | . | " generally irregular. | | | | | |

(δ.) In *quiet* and considerably demented general paralytics the conditions were:—

| | | | | | | | |
|---|---------------------|---|-----------------|---------------------|---|--|--|
| Symmetry | . | <table border="0"> <tr> <td>Pupils equal.</td> <td rowspan="4">} in equal numbers.</td> </tr> <tr> <td>Right pupil <i>persistently</i> tending to become the larger.</td> </tr> <tr> <td>Left pupil <i>persistently</i> tending to become the larger.</td> </tr> <tr> <td>Pupils equal, or the left larger <i>at times</i>.</td> </tr> </table> | Pupils equal. | } in equal numbers. | Right pupil <i>persistently</i> tending to become the larger. | Left pupil <i>persistently</i> tending to become the larger. | Pupils equal, or the left larger <i>at times</i> . |
| Pupils equal. | } in equal numbers. | | | | | | |
| Right pupil <i>persistently</i> tending to become the larger. | | | | | | | |
| Left pupil <i>persistently</i> tending to become the larger. | | | | | | | |
| Pupils equal, or the left larger <i>at times</i> . | | | | | | | |
| Size | . | <table border="0"> <tr> <td>" " " right " "</td> <td rowspan="4">} in equal numbers.</td> </tr> <tr> <td>Pupils rather small.</td> </tr> <tr> <td>" about natural size (relatively to light).</td> </tr> <tr> <td>" dilated, both, or one only.</td> </tr> </table> | " " " right " " | } in equal numbers. | Pupils rather small. | " about natural size (relatively to light). | " dilated, both, or one only. |
| " " " right " " | } in equal numbers. | | | | | | |
| Pupils rather small. | | | | | | | |
| " about natural size (relatively to light). | | | | | | | |
| " dilated, both, or one only. | | | | | | | |
| | | " small. | | | | | |

| | | | |
|----------|---|---|---------------------|
| Mobility | . | Pupils, a mutable degree of sluggishness. | } in equal numbers. |
| | | " somewhat sluggish. | |
| | | " moderately sluggish. | |
| Shape | . | " very sluggish. | |
| | | " both irregular. | |
| | | " mutably irregular. | |
| | | " right or left alone irregular. | |

(c.) In extreme dementia, or amentia, of general paralysis.

| | | | |
|----------|---|--|---------------------|
| Symmetry | . | Right pupil persistently tending to become the larger. | } in equal numbers. |
| | | Left pupil persistently tending to become the larger. | |
| | | Pupils, equal. | |
| Size | . | " equal, or right pupil the larger <i>at times</i> . | |
| | | " Pupils, about natural size (relatively to light). | |
| | | " slightly contracted. | |
| Mobility | . | " either right, or left, pupil, or both, dilated. | |
| | | " very sluggish. | |
| | | " somewhat sluggish. | |
| Shape | . | " mutably sluggish. | |
| | | " both irregular. | |
| | | " mutably irregular. | |
| | | " right alone irregular. | |

In reference to the above summary it may be noted that in a given eye, and under the same amount of light, the pupillary size and shape and the degree of iridal sluggishness may vary much from day to day, or even from hour to hour, the changes sometimes being very rapid. Also, that on the whole, and even in the later periods of general paralysis, I have found less pupillary dilatation than have several observers who made a special study of this subject. Nevertheless pupillary dilatation is a frequent condition.

After unilateral convulsions in general paralysis the pupil on the opposite side is usually, but not always, dilated for a time. I have often seen the pupils dilate at the commencement of the epileptiform seizures.

At an early date Moreau (de Tours) supplemented, by other facts, the discovery of pupillary inequality in general paralysis made by Baillarger. In 100 of his patients at the Bicêtre Moreau* observed inequality of the pupils in fifty-eight p.c.; the pupils in twenty-four p.c. being greater on the right; in thirty-four p.c. greater on the left, side; increased convexity of the ocular globe in sixty-six p.c.; odd shape of the eyebrows, which were separated at the inner end, or rose or fell abnormally

* *L'Union Médicale*, No. 78, T. vii. July 2, 1853, p. 310.

from the middle of the arch, in fifty-one p.c.; and often also a bluish sclerotic tunic, long upper eyelashes, and short, thin lower ones.

The contributions of Mobèche* contain a detailed consideration of the ocular, and especially of the pupillary, conditions in general paralysis.

Twenty years ago the sweeping generalization was made by Austin† that when the patient was depressed and melancholic the right pupil mainly or almost solely was affected; and that the left was the one principally involved when the patient was maniacal and the subject of exalted delusions.—Also, that the rule was confirmed by the variations of the unsymmetrical pupils. Thus:—a depressed general paralytic becomes more melancholic, his slightly dilated right pupil dilates still more; he improves, the dilatation recedes:—and similarly with the exaltation of the exalted general paralytic and the left pupil. Finally, that the rule was confirmed in those general paralytics whose pupils are usually symmetrical, but who have sudden accessions of melancholy or of exaltation. I am not aware that these statements have been confirmed.

Deviations from the usual contracted condition of the pupils during sleep were found by W. Sander‡ in general paralysis. Either the contraction did not take place, or a dilated pupil dilated still more during sleep, that of the other eye contracting as much as, or less than, usual; or a pupil, contracted during the waking state, became dilated as sleep set in.

Ophthalmoscopic Appearances. — Marked ophthalmoscopic changes in general paralysis have been seen, more especially by Koestl and Mimetscheck,§ Clifford Allbutt, and C. Aldridge. In some few cases they were observed by Westphal and Graefe, while Voisin and Galezowski but rarely found them. Voisin,|| however, has observed aneurismal dilatation and general dilatation and tortuosity of the central artery of the retina, as well as a blocking of it. In most cases Magnan traced changes along the central arteries of the retina.

It is stated by Clifford Allbutt¶ that atrophy of the optic

* *Annales Médico-Psychologiques*, Nov. 1874, p. 325; Jan. 1875, p. 19. On this subject see, also, Laseque, "Thèse d'agrégation," 1853.—Austin, *Op. cit.*, 1859.—Marcé, "Traité des Maladies Mentales," 1862.—Billod, *Ann. Méd.-Psych.*, 1863.—Voisin, *L'Union Médicale*, 1868.

† *Op. cit.*, p. 34.

‡ "Archiv für Psychiatrie und Nervenkrankheiten," ix. Band, 1 Heft, p. 129.

§ Quoted by Voisin, *L'Union Médicale*, Aug. 4, 1868, p. 180.

|| *Ibid.*

¶ "On the Use of the Ophthalmoscope," &c., London, 1871, p. 393.

nerve occurred in almost every case of general paralysis, the atrophy attacking them primarily, and not descending from the optic centres, and often becoming apparent as a hyperæmia with slight exudation, and followed by whitening of the disc proceeding from the outer edge inwards, the nerve becoming white and staring, or occasionally of slate colour, and its edge sharply defined. Moreover, he concluded that any proportional relations of the atrophy were rather with the pupillary states than with any ataxy of the orbital muscles.

That the affection of the optic discs in general paralysis commences by inflammation and slight exudation, and ends in atrophy, was observed by C. Aldridge.* Some discs were pink and hazy, others deep hazy-red and slightly swollen, others white with a faint capillary tint, and, lastly, some quite white and atrophic. "The disease generally ends by one side of the disc, usually the inner, becoming white and atrophic whilst the inflammatory changes are still in progress at the other. In some cases, however, the disc has a white rim, and a very large and shallow excavation of an extremely pearly-white tint occupies the centre, the remaining portion being of a greyish pink tinge." The atrophy of the disc was most complete in female cases, and was most advanced in the left eye, as a rule.

CHAPTER III.

FURTHER CONSIDERATIONS OF THE COURSE OF GENERAL PARALYSIS.

We may here resume some further consideration of the *course* of the disease.

A. Precedence of Orders of Symptoms.

Much discussion has arisen as to which in general paralysis precede the others, the *motor*, or the *mental*, symptoms. We need scarcely more than glance at the views of several authorities on this subject.

Bayle admitted that the affection of speech frequently preceded any mental alienation, and added that most often it followed

* West Riding Asylum Reports, vol. ii. p. 223.

an attack of congestion. That the mental almost invariably preceded the motor symptoms was the view of Delaye, a view very similar to that of Esquirol, Rodriguez, Requin, Griesinger, Conolly and Jules Falret.

In 1826 Calmeil wrote that the motor symptoms sometimes followed the mental; sometimes came on at the same time; rarely, preceded them; and in 1859 he confirmed this statement from his large intermediate experience. Much to the same effect were the expressions of Foville (*père*), and of Maudsley, and by 1838 Esquirol had so far modified his earlier views as to allow that the "paralysis" *did* sometimes precede, or occur simultaneously with, the outburst of mental alienation.

Again the usual simultaneity of the two orders of symptoms was asserted by Daveau, Broussais and others.

But Baillarger held that in general paralysis the "paralysis" was the primitive and principal element, which preceded the insanity in most, or all, cases, the insanity, indeed, being only secondary and accidental, and when present really a complication of the paralysis. Guislain, Lunier, Requin, Prus, Duhamel acknowledged this, at least for some cases; and in others Guislain asserted that general paralysis may begin with abolition of speech, while the late David Skae had occasionally seen the affection of speech and gait precede the insanity by a long period, and Leidesdorf reported a case in which the first symptoms were spinal and motor.

Of Parchappe's cases the mental and motor symptoms came on simultaneously in the majority (51); but the mental preceded the motor in a goodly number (27); while the motor were not proved to precede in any case; the order of precedence not being ascertained in 8. Compare with these the statistics of Brierre de Boismont:—disorder of intelligence and of motility coming on simultaneously in 34 cases; the mental symptoms preceding in 42 cases; and the motor symptoms preceding in 16. Or, adopting a tabular form:—

| — | Mental and Motor Symptoms occurring simultaneously, | Mental Symptoms occurring first, | Motor Symptoms occurring first, |
|-----------------------------------|---|----------------------------------|---------------------------------|
| Of 78 cases, Parchappe found ... | in 51 | in 27 | in 0 |
| Of 92 cases, Brierre found | in 34 | in 42 | in 16 |

Considerations of the Course of General Paralysis. 59

By which is seen at a glance how different were the relative proportions assigned to the three classes by these observers.

Intimately allied with this is the question whether general paralysis ever supervenes in old cases of ordinary "neuropathic" insanity.

Delaye spoke of the motor symptoms as sometimes supervening in old cases of insanity, and Conolly wrote that "it often happens that general paralysis supervenes after years of insanity in cases already hopeless, and it is then merely the beginning of a sure though slow decay." Jules Falret, in giving the pre-eminence to the mental derangement, at the same time denied that the "paralysis" ever came on in old cases of insanity, and Guislain had never seen ordinary mania, melancholia, or dementia pass into general paralysis, although he admitted that it might occur as an incidental termination of congestionary delirious mania. But Parchappe believed that he sometimes found general paralysis supervene on ordinary chronic insanity. No modern writer, however, as far as I know, avows the last and untenable portion of the bold generalization of Broussais "that all partial insanities tend to become general, and the general tend more or less to dementia and *general paralysis*." The criticism refers only to the words italicised here, but not italicised in the original.

Undoubtedly many patients are insane for some time, occasionally for years, before any motor indications can be detected even by close examination. I have noticed the somatic signs supervene weeks; months; a year and a half; between two and three years; after the commencement of highly pronounced mental alienation. I speak now of cases in which general paralysis was suspected and its physical indications carefully watched for. The primary mental alienation was of various forms: acute delirious mania;—acute mania;—expansive delirium;—intermingled hypochondria and melancholia with agitation;—dementia. In these cases, is the primary mental alienation but a phase of "general paralysis" in the psychical sphere, or is it independent of the latter? A question difficult to resolve. In his recent work, published since the above was written, Voisin adopts a factitious division of these cases. He makes a duration of two years the criterion. For him, mental alienation, if followed by the somatic signs in less than two years, pertains to "general paralysis," but if more than two years have elapsed ere the somatic signs appear, then the primary alienation pertains to simple insanity. He also admits of a remission of not more than two years in the instances pertaining to general paralysis.

I should prefer to adopt the form and characteristics of the

60 *Sequence of Symptoms in General Paralysis.*

mental derangement as a criterion. Usually some of the mental traits have suggested general paralysis ere its somatic signs appear.

B. The Mode and Time of Sequence of Individual Symptoms in General Paralysis.

As to the mode and time of sequence of the individual symptoms, only the most general outlines can be indicated here. As Jules Falret said, there is usually a general progression in the intensity of the symptoms both physical and moral, and on the other hand irregularity in the degree and order of appearance of the different phenomena. Moreover, there is often much fluctuation in the course of the mental symptoms, and even of the motor; nor can we agree with Esquirol when he says that the march of the paralysis is unceasing and that it always continues to increase.

In the motor sphere there are in general terms and in order of time:—I. ataxy; inco-ordination; II. paresis added to ataxy; and, III. increase of ataxy and of paresis to a state of helplessness.

More particularly; the motor affection of the apparatus of speech is usually that first recognized; and, together with the tremor and twitching of the tongue, lips and face, the inco-ordination of the locomotor movements of the lower limbs is often noticed early in the disease. Ocular modifications, and impaired co-ordination of the finer movements of the upper extremities, may soon be observed, or occasionally may even appear to precede the labio-lingual disorder.

The word "paralysis" is immediately about to be used in deference to the writers quoted, but the motor impairment in question is an ataxia and not a paralysis. According to Foville (*père*) the "paralysis" begins in the tongue, and Belhomme stated that impairment of speech preceded any other paralytic symptoms, and that the lower limbs were affected before the upper, while, on the contrary, according to Rodriguez and Brierre de Boismont the paralysis affected the upper before the lower extremities. Doutrebente concluded that when the cause is sexual excess the paralysis often begins in the lower extremities and travels upwards, and I have seen at least one clear example of the association of this causation and course. D. Skae found that speech alone (10 cases), or the gait alone (8 cases), may be affected, or, more usually, both (90 cases).

Much discussion has arisen with regard to these points, but, whether it explains the disagreement of authorities or not, there

seems to be little doubt that the motor disorder affects different parts of the system first in different cases.

Nevertheless, it is held by some that the general voluntary muscular system is uniformly affected, but that the impairment of the *speech* and of the *gait* is rendered more obvious than that of other motor activities because of the complexity and exactitude of the muscular co-ordinations and harmonious adaptations required in the one case; and the force and sustained effort necessary in the other.

As to the *mental symptoms* in this relation, one cannot, with Bayle, divide general paralysis somewhat rigidly into three successive periods, namely, those of; I. ambitious monomania;—II. mania;—and III. dementia. The mental symptoms occur in the most varied possible time and mode of sequence. It need only be said that the exaltation and extravagant notions, if present, are, as a rule, most active at, and immediately after, the onset; that the more active maniacal symptoms may occur then or somewhat later, or even intervene in the period of extreme dementia; and that the hypochondriacal or melancholic symptoms may be prominent almost throughout the course of the affection, yet occur more particularly in the middle periods, occasionally run into the later periods, and now and then are found at the onset, especially the melancholic. Dementia, which occasionally is predominant, or even exclusive, from the outset, is present, more or less, in all cases from the early stages, is irregularly progressive, and is often very extreme towards the close of life.

The emotional state often tends at first to be gay and expansive; later, to be depressed, morose or peevish; and, finally, to be reduced to the vanishing point.

The moral qualities, perturbed at first and inciting to irregular actions as if from moral turpitude, rapidly undergo disintegration and decay.

The sensory functions, both general and special, become blunted or even lost, but less so than other functions, and, as a rule, only somewhat late in the course of the disease.

C. The Relative Degrees of Intensity of the several Symptoms.

In the motor sphere, speech and locomotion are almost invariably the functions most obviously affected; the lower limbs *apparently* suffering more than the upper. But the contention as to this has already been noticed.

In the mental sphere, exalted delusion is a very striking pheno-

menon, so, too, is the hypochondriacal, or occasionally the melancholic, condition; and towards the last dementia is often so intense as to overpass the bounds of amentia. The disintegration and destruction of the mental powers are the ever-present and essential psychical conditions.

In general terms, both the mental and motor symptoms are throughout more intense, as a rule, than are the sensory; and the mental derangement may run riot at a time when the motor is scarcely discernible. Yet during remissions the mental symptoms often clear up much more than do the physical, and, indeed, may disappear altogether for a time. Now, to say the very least, this entire disappearance is much more rare in the case of the physical signs. (See also the section on remissions in general paralysis, *postea*.)

D. The Question of the Existence of General Paralysis without Mental Alienation.

Can general paralysis exist and run its course without mental symptoms, the disease in other respects being that called "general paralysis of the insane"? Oft-repeated and animated the discussion, and conflicting the views on this question.

The possibility of this absence of mental symptoms was at first denied by Calmeil, and a third of a century later he maintained that it only occurred with extreme rarity, if at all. The earliest recorded case supposed of general paralysis without mental symptoms—namely, that by Delaye—and some, at least, of Lunier's cases were not, I believe, true instances of general paralysis. Baillarger, who stated that it might exist without *délire* and with some dementia only, called general paralysis a special disease, independent of insanity, in which the lesions of motility appear first, and the mental symptoms may be absent. Guislain and D. Skae had seen it with some loss of memory only, and W. T. Gairdner recently exhibited two cases resembling general paralysis but without insanity. Copland asserted that he had observed cases of the same import.

I think if cases of this kind are followed up it will usually be found that they resolve themselves into the form of general paralysis with dementia only, in the psychical sphere; in some of which, perhaps, the extent of departure from the more usual and more dramatic form is widened by the relatively early, and relatively predominant, ataxy and disabling paralysis or paresis.

This subject cannot be further pursued in the present place. Next to the question of their very existence, it has principally been in dispute whether (if they exist) the cases without mental

alienation constitute a distinct disease, or are really the same as "general paralysis of the insane" except for the omission of the mental symptoms, which last, in this view, are said to be non-essential. That such cases constitute a distinct disease was believed by Duchenne and Brierre de Boismont because of the absence in them of encephalic lesion, and because of the abolition of electro-muscular contractility. If really a distinct affection it evidently constitutes a third disease; distinct from general paralysis of the insane on the one hand; and, on the other, distinct also from those more or less generalized *palsies* which may follow various ordinary pathological changes in the nervous system.

REMISSIONS IN GENERAL PARALYSIS.—The remissions to which the disease is occasionally subject were described by A. Sauze* as of three kinds. Of these it is not uncommon to see two; namely, that in which there is remission of the mental and motor symptoms simultaneously; and that in which the remission pertains to the mental symptoms only, or chiefly. I have never seen clear decisive examples of the other variety mentioned by him; that is to say, decisive remission of the motor symptoms alone, the mental remaining without improvement, although a slighter degree of this is not rare. On the contrary, the little I have seen for the most part coincides with the experience of Baillarger,† who long ago spoke of a dissociation of the two orders of symptoms, of such sort that the insanity disappears—the generalized paresis persisting—and who referred to similar cases by Bayle, Ferrus and Rodriguez.

It has recently‡ been asserted by Baillarger that its remissions are most frequent when the general paralysis begins with a maniacal attack;—easily explained, if true, on the hypothesis that the various forms of simple insanity often complicate general paralysis at its onset, of which forms mania is one most likely to terminate in recovery. According to this view§ the form in which there is dementia only, is in reality general paralysis with its essential symptoms—those which never fail—and in this light, also, it is impossible to view the attacks of mania, melancholia, or monomania with which the disease sometimes comes on, otherwise than as complications;—and general paralysis, again, usually a primary affection, as sometimes being secondary, in a sense.

Luchs|| declares that remissions in general paralysis are merely

* *Ann. Méd.-Psych.*, Oct. 1858.

† *Ibid.* May, 1847, p. 335.

‡ *Ibid.* May, 1876, p. 256.

§ *Ibid.* p. 263.

|| *Société Méd.-Psych.*, May, 1877; *Ann. Méd.-Psych.*, July, 1877, p. 111.

due to the cessation of epiphenomena, the fundamental lesions, nevertheless, persisting and progressing.

On the contrary, Douthett* argues for the complete and lasting character of certain of the remissions, or, in other words, the recovery of the patients. And this most frequently in the maniacal or acute form of general paralysis; while long remissions occurring in chronic general paralysis, fall rather to the share of patients with an hereditary neurotic predisposition. Embarrassment of speech he found to be the most tenacious symptom during the remissions. Baillarger,† however, has collected or described several well-pronounced instances of remission, and possibly of recovery, in general paralysis of the form in which the mental symptoms consist of *melancolie avec stupeur*, or of *stupeur*. Here, as in the ordinary expansive, maniacal, or melancholic, forms there is, for him, not a true dementia, but a special state—one of *pseudo-dementia*—analogous to that of certain examples of drunkenness, and permitting of decisive remissions. When the dementia of general paralysis is slow, progressive, simple, *i.e.* unlinked with any mental disorder, then the dementia, in these comparatively rare cases, is real, and, like that of ordinary chronic secondary neuropathic dementia, is incompatible with the occurrence of any remissions worthy the name. These remissions in the more *usual* dementia of general paralysis were long ago made a principal point in its distinction from ordinary chronic mania by W. Wood.‡

A gentleman, now under my care, is said to have had a temporary attack with maniacal symptoms above four years ago. More than three years ago he first came under my care with acute maniacal agitation, expansive delirium, and *masked* physical signs of general paralysis, the diagnosis of which, made with some difficulty, was justified by the subsequent manifestation of slight somatic signs. Complete remission or disappearance of the mental symptoms took place, except of the incapacity for steady application to work; and incomplete remission of the physical. He was discharged, remained fairly well for a time, but was readmitted eight months afterwards suffering from general paralysis; on this occasion with less active maniacal symptoms; again a very decisive remission occurred; he was again discharged, and on this occasion remained pretty well for nearly a year, at the expiration of which period he was admitted a third time, and now with active maniacal symptoms and

* *Ann. Méd.-Psych.*, 1878, March, p. 161; May, p. 321.

† *Ibid.* Jan. 1879, p. 5.

‡ *British and Foreign Medico-Chirurgical Review*, July, 1860, p. 199.

ambitious delirium, the mental powers also being shattered, and the physical signs of general paralysis highly marked and characteristic. From this state he has at the moment of this writing again made considerable recovery, especially in the mental sphere.

A much more remarkable case of this kind has recently been placed on record by Lunier,* in which the attacks and remissions or recoveries, seven in number, extended over a period of twenty-three years.

Other cases of prolonged remission, in my own practice, will be referred to hereafter when speaking of the *prognosis*.

The remissions referred to in this section are not mere points of sinking in the ordinary fluctuations of the disease, but are of decisive nature and persist for a time, as, for example, six months in a case reported by N. G. Mercer,† or in that of Bonnefoust‡ lasting four years. Of twenty cases of remission mentioned by Baillarger the two longest were of about two years' duration, and of the twelve cases recently published by Doutrebente the longest complete (?) remission endured two years and two months. This followed an attack of pleurisy, but some intellectual debility remained.

Without any remission the lethal course of the disease may also be simply arrested for months or even years, the mental and motor symptoms, moderate in degree, being apparently stationary during that period.

CHAPTER IV.

THE DURATION.

THE duration of general paralysis varies from a few weeks or months to one, two, or several years. Aside from lethal, independent and intercurrent accident or disease, death may be brought about in the early periods either by the severity and extent of the morbid process in the nervous system, or by incidental cerebro-spinal or visceral complications.

* *Ann. Méd.-Psych.*, March, 1879, p. 239.

† *Medical Times and Gazette*, Dec. 1867, p. 617.

‡ *Ann. Méd.-Psych.*, May, 1869, p. 433.

In 1826 Bayle estimated the *average duration* of general paralysis at from one year to one year and a half, while at the same date Calmeil stated it as being thirteen months, and in his later classical work he asserts that at one period Bayle had fixed it at ten months. In these figures both the male and female cases are included. On a basis of forty-two cases Parchappe* estimated the average duration at twenty-three months. He found the most rapid cases to be those in which the delirium was like that of the various acute forms of simple insanity, and also those in which the "paralytic" signs were observed from the outset; while the longest cases were those in which the "paralytic" signs appeared later than the mental disease, and those in which the latter was of the nature of dementia only. At the Wakefield Asylum† the male cases of general paralysis during a series of years were of the *average duration* of 20·7 months; the female, of 25·9 months: total average duration, male and female, 21·6 months. At the Devon County Asylum‡ the average duration was; males, 15 months; females, 27 months; total average, 21 months.

If sufficiently large numbers are examined, the *average duration* is always less in male than in female general paralytics. This is merely a fact in the natural history of the disease, although its validity as such has been ignored,§ and the fact, in part distorted from its true meaning, made the basis of an eulogy on the nursing apportioned to the females.

Nor, with respect to duration, does the natural history of the disease include only the difference as to the sexes. Not only is general paralysis a more rapid disease, on the average, in the male sex, the one in which it is more rife, but it is more rapid on the average, also, in patients of those classes of society, or following the occupations, or given to the habits or excesses, which render them prone to general paralysis. Hence we find the longest *average duration* of general paralysis in gentlewomen; the next, in men of the upper and middle classes, or in women of the lower orders, and the shortest in males of the lower orders; while among miners, ironworkers, soldiers, and others notoriously liable to general paralysis one expects to find the shortest average duration of all.

Speaking now of male cases only: among the gentlemen-

* "Recherches sur l'Encéphale, sa Structure, ses Fonctions, et ses Maladies," Paris, 1836, p. 155.

† West Riding Asylum Medical Reports, vol. v. p. 202.

‡ Ibid. vol. i. p. 138.

§ Ibid. vol. i. p. 139.

patients under my care some cases of general paralysis have been most protracted, as, for example, more than six, and ten years, and in them the *average duration* is very much higher than that found in the soldiers also under my care. The average duration in those soldiers upon whom it has chanced that I have made necropsies has been more than twenty-two and a half months; but adding the others, who are either already dead; or who, temporarily improved, were discharged; as well as the general paralytics who form the still living residue of those admitted here; the *average duration* is more than two years. This is a *high average duration* for *male* cases drawn from the *lower* orders of society as these soldiers are.

TERMINATION.—Though recovery is possible from its less advanced degrees, yet the termination of general paralysis is almost invariably in death. The patient, lying bedridden, with exhausted attenuated frame the prominences of which lose their vitality under the mere pressure of body-weight,—or parts of which melt down more rapidly or slough under acute trophic disorder of cerebral or of spinal origin—this patient, I say, may be cut off by the ulterior effects of these lesions,—effects such as exhaustion, septicæmia, pyæmia, ichorous spinal meningitis, infarction and gangrene of lung;—or, again, may succumb under other pulmonary or abdominal diseases, so frequently accompanying general paralysis, particularly hypostatic pneumonia, diarrhœa, nephritis and cystitis;—or may be cut off by such incidents or complications as intracranial (or intraspinal?) hæmorrhage, or severe epileptiform convulsions, or embolism; or, the throat-power failing, may die choked by food, or, more slowly, from pulmonitis sequential to its inhalation. In some cases no new symptoms are set up; death is brought about by gradual exhaustion, apparently beginning in the cerebro-spinal system.

The prolonged remissions already described are not altogether rare; and, again, the rapidity of the lethal descent may be stayed, and for a time the case may proceed on the level lines of a chronic state of dementia and ataxia; but neither of these can truly be called terminations of the disease.

Including both sexes, of the deaths occurring in 36 British asylums, in a certain year (1869), $18\frac{1}{2}$ *per cent.* were attributed to general paralysis;* and of the deaths during a quarter of a century at the Devon County Asylum† $29\cdot25$ *per cent.* of those amongst the *males*; and $7\cdot37$ *per cent.* of those amongst the *females*, were from general paralysis; or, taking both sexes,

* West Biding Asylum Reports, vol. i. pp. 131 and 130. † Ibid.

19.45 per cent. of the total number of deaths were those of general paralytics.

Among the soldiers, only, under my care, of 238 deaths 70, or 29 per cent., were due to general paralysis, with or without complications.

CHAPTER V.

DIAGNOSIS.

In the well-marked cases the diagnosis is unattended by difficulty, but there are many instances in which it is far from easy; or, at some periods, is impossible. The physical signs are those most relied upon, especially the characters of the speech, the fibrillary trembling and twitching of the muscles of the tongue, lips, and face when in use, the similar ataxia of the extremities, and the pupillary changes. The sensorial, spasmodic, convulsive, parietic, and paralytic phenomena are of help, when present.

The differential diagnosis must be established between general paralysis and a number of affections.

1. *Chronic Alcoholism*.—Chronic alcoholic intoxication is often the exciting cause of general paralysis, but it is necessary to separate the conditions usually termed "chronic alcoholism," from general paralysis by whatever cause produced.

A case recently admitted under my care when of one month's duration, and in which the cause of insanity was stated to be unknown, resembled general paralysis very closely both in its mental and physical symptoms. But the diagnosis arrived at—namely, chronic alcoholism—was justified by the complete recovery of the patient, and was based upon the following points:—(a) The general tremulous condition, and the marked tremors of the face and hands at so early a period.—(b) The constant, restless, fumbling, busy movements.—(c) The incomplete development of the affection of speech as compared with that usual to well-marked general paralysis.—(d) The age of the patient (23).

Nor was his admission of alcoholic excess entirely without significance.

Yet was there a great resemblance to general paralysis. There

were constant mental restlessness and confusion, and large and extravagant notions, though without much accompanying exaltation of feeling. On the negative side, again, there had been no morning vomiting, and no evidence of hallucinations was manifest, nor were there positive feelings of dread, nor red eyes, nor acne rosacea, nor flabbiness of features at the very onset, though these last had lost in part their natural mobility;—while no tiinitus aurium, no clouds or flashes of light before the eyes, no vertigo, and no hideous nightmare were complained of. Hence a dissidence from the more ordinary features of chronic alcoholism.

Fortunately, in the majority of cases the resemblance is not so close. Usually it is the general paralysis characterized by depressive or melancholic symptoms to which some cases of chronic alcoholism, or alcoholic mania, bear so strong a resemblance.

In chronic simple insanity from alcoholic excess the affection of speech is usually less than in general paralysis, and is proportional to the amount of tremor present; the disorders of special, and the failure of general, sensibility, as exemplified in headache hallucinations anæsthesiæ and pricking sensations, are more marked in it, on the whole, though, on the other hand, Voisin considers loss of smell far more frequent in the general paralytic than in the alcoholic case;—the hallucinations and delusions of chronic alcoholic mania, also, are ordinarily of a special character and are such as delusions of persecution, fear, suspicion, with self-abasement, sombre feeling, or tendency to suicide; while, with less incoordination and less frequent pupillary changes, there is more of general muscular tremulousness in it than in general paralysis; and gastric catarrh, nocturnal disquietude, morning tremor, and brutish expression are frequent symptoms. The old drunkard, too, is more apt to complain bitterly of his sufferings.*

Long ago Ch. Lasèque† indicated that the alcoholic trembling was universal, and without points of election as in general paralysis;—that at the early stages of each the muscular feebleness in alcoholism was in contrast with the spasmodic muscular incitations in general paralysis, and that in the former both the contentment and the indifference of general paralysis were wanting.

* See Léon Thomeuf on the differential diagnosis of alcoholic lypemania complicated with paralytic accidents, and general paralysis.—*Gaz. des Hôpitaux*, July 19, 1859, p. 334.—*Ann. Méd.-Psych.*, 1859, p. 564.

† “De la paralysie générale progressive.” Thèse, 1853.

But when general paralysis arises from alcoholic excesses the diagnosis may be extremely difficult, especially if, as may happen, in these cases there are symptoms such as visual hallucinations, delusions of persecution and of being poisoned, and, perhaps, homicidal and suicidal tendencies.

Dr. Batty Tuke* looks upon local colour-blindness, and hyperæmic retina, with a contracted irregular pupil, as important symptoms in general paralysis, distinguishing it from chronic alcoholic mania. In the latter, however, hyperæmia of the retina may also be found, but it subsides when stimulants are cut off.

Acute alcoholic mania sometimes resembles general paralysis of the maniacal form, and on occasions I have found that final judgment must be suspended for a short period. In a case of this kind Batty Tuke found all the symptoms of general paralysis except the alterations of the pupils.

2. *Syphilitic Disease of Brain and Meninges.*—In the *British and Foreign Medico-Chirurgical Review* for April, 1877, I have treated at great length of the differential diagnosis between general paralysis and certain cases of intra-cranial syphilis, and therefore will only refer briefly to it here.

(A). The semeiological form of general paralysis most frequently simulated by intra-cranial syphilis is that in which dementia is the principal mental symptom from the first, the exaltation and the ideas of grandeur being absent or evanescent, while there may be a tinge of depression, with occasional or frequent fear or terror.

On comparing its *mode of onset and earlier symptoms* with those of the form of intra-cranial syphilis which simulates it, we find, as I have said in the paper just referred to, that “while there is much that is similar in the syphilitic cases, yet, on the other hand, we may find in them an early hypochondria or temporary acute mania or delirium, and the mental symptoms are often *preceded* by marked motor or sensory disorders. Early paralysis of some of the cranial nerves with ocular troubles, early optic neuritis, or local anæsthesiæ, often characterize the syphilitic cases. Headache, nocturnal, deeply seated, and increased by pressure or warmth, is usually a striking phenomenon, and is more urgent and persistent than is the prodromal headache observed in some cases of general paralysis. Convulsions and local spasms are more frequent in the first periods than in the

* *British Medical Journal*, 1877, vol. i. p. 744.

corresponding periods of general paralysis. Early insomnia, common in some instances to both, is, as a rule, more severe in the syphilitic cases, and the same remark applies to rheumatoid pains in the extremities and to the various neuralgiæ at the same period. A fitful appearance, and capricious association and succession of the several symptoms, and their frequent alternations, are features more evident in the syphilitic cases. Anæmia is frequent, and a sallow cachectic hue, not usual to general paralytics at this period. The early or rapid decline of memory, sometimes insisted upon, is, however, common to both; nor is there any very marked difference between the occasional early convulsive phenomena of the two diseases, which in both are commonly unilateral, and often without loss of consciousness. The impaired articulation, and tremors and twitchings of lips, face, and tongue may be absent or but imperfectly marked; but, on the other hand, they may be imperfectly marked in general paralysis (its *dementia* form) also. *Early* well-marked apoplectiform attacks, severe nocturnal cranial pain, paralysis of individual cranial nerves, local muscular contraction and rigidity, optic neuritis, vertigo, early failure of special senses, and local anæsthesiæ, are the most important distinguishing features of intra-cranial syphilitic cases in the incipient stages; and these, when present, greatly facilitate the differential diagnosis between the particular class of cases of intra-cranial syphilis now under discussion and general paralysis-dementia. On the other hand, the characteristic condition of the articulation, facial and labial muscles, tongue, velum and pharynx, when fully developed at this early period, often suffice to establish the diagnosis of general paralysis."*

In the next place the features of the same two affections when *fully developed*, are contrasted in the same article. (*a*). *Mental*. Certain differences in the mental symptoms are there detailed.† (*b*). *Physical*. Speech may be very similar in the two cases. "The great distinction, however, is that in the syphilitic cases the affection of speech, tongue, &c., when it exists, is almost always mainly of a *paralytic* nature, instead of being of the nature of a mingled weakness and incoordination."‡

Dysphagia is usually sudden in syphilis, gradual and progressive in general paralysis. In the general-paralysis cases are certain pupillary and ocular peculiarities, and often atrophy of

* *British and Foreign Medico-Chirurgical Review*, April, 1877, p. 448.

† *Ibid.* p. 450.

‡ *Ibid.* p. 451. These, and many other points, are detailed in the same paper.

the optic discs, after slight inflammation; in the syphilitic are often extreme double optic neuritis, disseminated choroiditis, and more frequent and more sudden blindness.

Characteristic of syphilis in this relation are palsies of individual cranial nerves: these are often complete, strictly limited, and independent of convulsive action. In general paralysis, on the other hand, sudden local palsies or monoplegiæ usually follow local spasm or convulsion, are incomplete, and more often are transitory and recurring. The electrical reactions differ here also. In the syphilitic cases the muscles of the palsied part often show abolition of farado-contractility, and exaggerated reaction to the intermitted constant current.

The customary motor impairment in *the limbs* and other parts in general paralysis is ataxic, and (usually also) paretic, is general, and, for the most part, irregularly progressive: in syphilis it is usually *paralytic*, localized, and unilateral, though sometimes general, and may be stationary or retrogressive;—and palsy of one or more cranial nerves, convulsions, or anæsthesiæ often co-exist.

Loss of special senses occurs more frequently, and often more rapidly or suddenly, in the syphilitic cases. Cutaneous "anæsthesia is often early and local in the syphilitic; late, general, and progressive in the general paralytic" (*loc. cit.* p. 445); but to these there are exceptions.

Severe cephalalgia, worse at night, and general osteocopic pains often afflict the syphilitic.

In syphilis apoplectiform seizures usually leave more marked traces than in general paralysis.

(B). Passing now to the expansive form of general paralysis and comparing it with the intra-cranial syphilitic cases most closely resembling it.

Maniacal attacks may occur in both cases, but some subsidiary points of contrast may usually be recognized; as, for example, in the maniacal attacks of general paralysis—the preceding premonitory and other symptoms, the attending emotional exaltation, and elevation of temperature, and the subsequent augmentation of the motor disorder and impairment. (*loc. cit.* p. 456.)

"But again a difficulty arises, inasmuch as in some cases of intra-cranial syphilis simulating general paralysis, extravagant notions and exalted delusions occur; while the gait is tottering, speech and writing are impaired, and there is an inability to dress,

and a blurring of the lines of facial expression, somewhat as in general paralysis. Without contrasting at length this form of syphilitic mental disease, and general paralysis with exaltation, and thereby recapitulating much that has been written above on the dementia form of general paralysis and the syphilitic disease most like *it*, we may at once say that when exaltation of feeling and exaggerated or extravagant notions arise in the syphilitic cases they are distinguished—

- a.* By the distinct history or symptoms of syphilis.
- b.* By the preceding cranial pains, nocturnal and intense.
- c.* The exaltation is less marked, less persistent, and perhaps less associated with general maniacal restlessness and excitement, than in most of the cases of general paralysis.
- d.* Sometimes by such complications as palsies of one or several cranial nerves, or hemiplegia, or paraplegia having the character and course of syphilitic palsies adverted to above.
- e.* By the greater frequency of optic neuritis, early amaurosis, deafness, local anæsthesiæ, vertigo, or local rigid contraction.
- f.* The affection of articulation is paralytic rather than paretic, and usually speech is not accompanied by any facial or labial tremors or twitchings.
- g.* By frank cerebral or spinal meningitis or pachymeningitis.
- h.* By the variety of the motor and sensory symptoms. . . .
- i.* By the effect of anti-syphilitic treatment.*

(C). *Mutatis mutandis*, very much the same holds true of cases of general paralysis taking the hypochondriacal and melancholic forms.

Dr. Leidesdorff has recently supported the views of those who aforetime laid stress upon the importance of localized indications of disease, such as palsy of a single cranial nerve, in cerebral syphilis.† Headache, spots of cutaneous anæsthesia, and palsy of single cerebral nerves are among the features specially relied on by Müller of Leutkirch.

A history of syphilitic infection, primary hypochondria, and, later, progressive intellectual failure, strongly indicates syphilitic

* *Loc. cit.*, pp. 456, 457;—one or two slight verbal alterations.

† Imp. Roy. Med. Soc., Vienna. *London Medical Record*, Jan. 1878, p. 41.

‡ Also Wille, Müller, Gros, Lancereaux, H. Jackson.

disease (Wille). The possibility of effecting a diagnosis may depend upon the occurrence of paralyzes of cranial nerves. Simultaneous and unilateral paralysis of the fifth and sixth cranial nerves is an almost certain indication of intra-cranial syphilis (Gräfe); the multiplicity of the nerve-trunks affected (Leudet); and the extension from nerve to nerve of the paralysis, and its sudden coming and going (Gros and Lancereaux); are striking and sufficiently characteristic traits. Yet they often fail us here.

According to Heubner* the cases, collected by himself, of intra-cranial syphilis bearing a close resemblance to general paralysis are precisely those in which no very decisive or characteristic anatomical changes have been visible to the naked eye. Yet in a case under Schüle, with gross intra-cranial syphilitic lesions there were microscopical changes in the blood-vessels and neuroglia of the cerebral cortex.

Nevertheless I† have shown that one kind of syphilitic brain disease which frequently, perhaps most frequently, simulates general paralysis is that in which the *cerebral arterioles* and, usually, arteries are extensively diseased, and in which sometimes also the cortical surface of the cerebrum and the overlying meninges are the sites of gummatous infiltrations. Adhesive meningitis is, I believe, another form of syphilitic disease that sometimes simulates general paralysis.

An apparent identity, in symptoms, with general paralysis was observed by A. Foville‡ in a case where the syphilitic lesion consisted of isolated gummy tumours, multiple, disseminated in the interior of the cerebral hemispheres, and only attaining the surface at a single narrowly limited spot.

Of cases mentioned by Zambaco§, in one the pia-mater was injected; the convolutions somewhat flattened; the peripheral grey matter softened, spotted, and detached with the pia-mater at several points; and the white central parts, such as the corpus callosum, softened. In another there was general softening of the grey substance, and a lactescent appearance of the arachnoid. In another the pia-mater was fibrous, thick, adherent, and apparently compressed some of the cranial nerves.

It has here been assumed that syphilis is not a *causa vera* of general paralysis. Many observers, however, have declared that

* "Ziemssen's Cyc. Trans.," vol. xii. p. 315.

† *British and Foreign Medico-Chirurgical Review*, July, 1876, and Apr. 1877; and *British Medical Journal*, July 13, 1878, p. 49.

‡ *Ann. Med.-Psych.*, May, 1879, p. 355.

§ "Des Affections Nerveuses Syphilitiques."

it sometimes stands in that relation, and the variance of opinion on the subject has been great.

On the one hand some, like Lancereaux,* while indicating that syphilitic encephalopathies may closely resemble general paralysis yet maintain that they are distinct and independent in character. So also Müller of Leutkirch, Buzzard, Fournier, and, to some extent, Wille.

On the other hand some maintain that syphilis may engender true "general paralysis of the insane." Among these are Jessen, Erlenmeyer, Ludwig Meyer, Westphal, Oedmansson, Griesinger and Wirze. Schüle, also, observed the typical symptoms of general paralysis produced by intra-cranial syphilis. Among them, also, is Rollet, who in support of this view quotes from the contributions of Richet, Follin, Melchior and Leidesdorf. Furthermore, Rollet summarized the symptoms and lesions of general paralysis due to syphilis: the symptoms, as being, progressive motor feebleness affecting all the muscles including the tongue, inability of the lower limbs to support the frame, trembling and uncertain movements of the hands, inability to grasp small objects properly, dysphagia, difficulty in speech, mental weakness and disorder. As for the lesions, he speaks of soft meninges resistant and whitish, adherent to the softened grey substance, of which a small layer separated along with the membranes; and, in some cases, of specific tumours, and of local softenings.

Whether, or not, syphilis may produce true general paralysis of the insane, it is unquestionable that in one class of cases syphilitic disease more or less closely resembles general paralysis in some of its features, and yet remains distinct therefrom in its essential nature. As between cases of this class and general paralysis, the distinctions drawn above will still prove serviceable, whatever be the ultimate fate of the major etiological question.

3. *Certain Cases of Acute Mania.*—Early in general paralysis there is sometimes a period of acute maniacal excitement, preceded by a stage of mental alteration, or of ambitious delirium, or by both, or by neither. In the opinion of some, "congestive insanity or mania" often is transformed into general paralysis, of which it then constitutes the first stage. One must, therefore, attempt to disentangle the active mania of general paralysis from certain cases of similarly active but simple mania.

In the cases already referred to, general paralysis can only be recognized by two orders of facts.

Firstly, by the existence of some slight hesitation, or occa-

* "Treatise on Syphilis," Trans. N.S.S., 1869, vol. ii. p. 66.

sional pause, or stumbling, in speech, or repetition, or partial repetition, or elision of syllables, with, possibly, a faint occasional twitching of the upper lip and face during speech and independently of any emotion, or slight tremulousness and twitching of the tongue on protrusion. (It is useless to speak here of hyperæmic retina and of failure of smell—both so difficult to test under these circumstances.)

And, secondly, by the predominant expression of extravagant ideas, and by the emotional exaltation.

But facts of both these orders are sometimes absent in mania which, without solution of continuity, subsequently merges with, or is transformed into, the psychological sphere of general paralysis: on the other hand facts of the former group are not unknown in ordinary acute mania, and those of the latter are not rare therein.

When the above somatic signs are absent or but doubtfully caught a well-founded diagnosis is impossible, and that, perhaps, for weeks or months. Again, and in other cases, when the physical indications of general paralysis are merely masked, as it were, the diagnosis cannot be made with certainty until the excitement subsides. As a rule this subsidence, or at least remission, in the maniacal excitement is not long delayed, but should it be tardy the diagnosis may remain doubtful. Under these circumstances mental confusion may be more marked in the general paralytic than in the maniac, and so may be any defect in writing, if the patient can be induced to write. Suspiciousness and morbid aversion or rage are more often absent, or transient and easily diverted in the general paralytic of this kind as compared with the ordinary maniac; while, on the other hand, a clue may be afforded by the occurrence of the epileptiform or apoplectiform seizures of general paralysis. The history of one or more previous attacks of mania or other form of insanity, would rather disavour the view that the case was one of general paralysis, but would by no means preclude it.

Expansive Delirium.—Mania, or monomania, of grandeurs wealth or pride often occur quite independently of general paralysis; indeed, the application of the latter term to certain mental symptoms of general paralysis is an usurpation. In doubtful cases of this sort Esquirol* had diagnosed general paralysis by an occasional slowness in pronunciation, and by the fact of the patient being calmed by a promise, and induced to forego apparently cherished projects. And it is particularly in cases such as these that the remarks of Esquirol apply:—that as compared with other, and ordinary, maniacs and monomaniacs, those with

* "Des Maladies Mentales," Paris, 1838, T. ii. p. 276.

(general) paralysis have not the same energy of attention, nor the same firmly-knit association of ideas, nor the same power of will, nor the same tenacity of resolve, nor the same obstinacy of resistance. Becoming excited and flying into passion yet do they yield and obey; but their acts already reveal the enfeeblement of the functions of the brain.

4. *Intra-cranial Tumours*.—The phenomena originated by intra-cranial tumour may to some extent simulate general paralysis and its epileptiform and apoplectiform attacks. But the tumour-cases are usually recognized by the existence of intense headache, vomiting, marked double optic neuritis, and failing sight; frequently, by deafness of one or both sides, or local cutaneous anæsthesia; sometimes, by local *complete* palsy due to compression of one or more motor nerves at the base of the brain—the paralyzed muscles showing diminished farado-contraction, and increased galvano-contraction—or, also, by other localized slowly progressive paralysis; by the absence of speech-affection, or by its truly paralytic, and not ataxic and paretic, nature, when present;—by more frequent rigidity, trailing gait, and vertigo:—and, occasionally, by the indications of tumours elsewhere or of special cachexiæ.

Yet the tumours may not be accompanied by these pronounced symptoms.

Especially does the difficulty in diagnosis occur in certain cases of cerebellar tumour in which there are found general impairment of muscular energy, a swaying staggering tottering unsafe gait, as well as vague and aimless movements, and, finally, perhaps, a want of control over the muscular movements—an incoordination—somewhat as exists in general paralysis. Other phases of general paralysis may be simulated by certain effects of the cerebellar tumour such as its pressure on the medulla oblongata, or general intra-cranial pressure by the ventricular effusion due to damming back of the blood-stream in the veins of Galen. Here the association of other symptoms usual in tumour—such as severe headache, vomiting, blindness—the difference in the gait and speech, and, almost always, in the mental symptoms must guide the diagnosis, and for this purpose the four most important symptoms are the occipital headache, rolling gait, vomiting, and visual failure or disorder.

In intra-cranial tumour, as compared with general paralysis, the mental symptoms are *comparatively* late, slight, or absent; the sensory comparatively prominent. The progressive dementia of certain cases of general paralysis, is simulated, not the grandiose delirium.

5. *Cerebro-Spinal Disseminated Sclerosis*.—Now and then a

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case of general paralysis in certain stages resembles cerebro-spinal disseminated sclerosis. A general paralytic may have the hands, or upper extremities, or head, or even the body at large, shaken by rhythmical tremor, associated with a general constant muscular restlessness; the tremor being markedly increased during any movement, or when the patient is in the erect position; and diminishing, or perhaps ceasing momentarily, when the parts affected are placed at rest from voluntary movements. There may also be a bending forward of the head and upper part of the body; the speech may be slow, deliberate, drawling, and the words measured and alternating with pauses. In all these respects, therefore, it may closely resemble the symptoms of disseminated sclerosis; while symptoms such as the following may be common to the two cases;—tremor of the tongue on protrusion, and of lips and face before and during speech, impaired and disordered lingual movements, disorder and impairment of visual power, uncertain embarrassed gait with the feet more widely apart than usual, and awkward clumsy movements, general and increasing paresis of the limbs, their final flexed contracture, irregular remissions and exacerbations of the various symptoms, and apoplecticiform or epileptiform attacks. In truth, there is a secondary miliary sclerosis in some cases of general paralysis.

Thus, in the one direction, is a close resemblance between the two affections reached by means of an approximation of the semeiography of general paralysis to that of insular sclerosis.

In the other direction, also, may the same result be arrived at, and insular sclerosis, in its turn, put on, in part, the garb of general paralysis. For the mental symptoms in disseminated cerebro-spinal sclerosis may resemble the dementia, or even the ambitious delirium, of different cases of general paralysis.*

When present, this simulation of the mental symptoms of general paralysis, especially of the expansive, vastly enhances the difficulties in the differential diagnosis. Here we can only rely upon the presence, or upon the greater frequency in disseminated

* Wilh. Valentiner, "Ueber die Sclerose des Gehirns und Rückenmarks." "Deutsche Klinik," 1856, Nos. 14, 15, and 16.

H. Liouville, *Mémoire lu à la Société de Biologie*, 1868. "Mém. and Comptes Rendus," p. 231. Especially the second case.

S. Jaccoud, "Traité de Pathologie Interne," 1869, T. i. p. 193.

Dr. Leubé, "Deutsch-Archiv," 8 Band, 1 Heft, Leipzig, 1870, quoted by Charcot.

J. M. Charcot, Course, at the Salpêtrière, of Lectures on the Diseases of the Nervous System. W. Moxon, *Guy's Hospital Reports*, vol. xxi. 1875. T. Buzzard, "Transactions of the Clinical Society of London," vol. viii. p. 121.

sclerosis, than in general paralysis, of such symptoms as temporary diplopia, nystagmus, vertigo, markedly staccato speech, and, finally, of "spinal epilepsy," so-called,* as well as of paroxysms of rigid extension of the lower limbs, and occasionally of the upper,—and ultimate permanent contracture,—usually in the position of extension, rarely in that of flexion.

As for the first-mentioned class of cases, that in which general paralysis counterfeits insular sclerosis, the diagnosis is aided by the impairment, in the latest stages of general paralysis, of the farado-contractility of the voluntary muscles especially in the lower limbs; whereas faradization of the latter in insular sclerosis usually brings on tetanic-like rigidity and convulsive trembling. The diagnosis is aided, also, by the fact that the usual early periods of general paralysis may already have been passed through without any simulation of disseminated sclerosis, as well as by the character of the tremor in these cases, which does not quite correspond with that of disseminated sclerosis; and, sometimes, by the more generalized paresis.

The comparative youth of many of the patients in cerebro-spinal disseminated sclerosis, its much greater *average* mean duration, and its greater frequency in females, are points of divergence from general paralysis, so also is the comparative lateness of any mental disorder or decay, which, indeed, is usually preceded by spinal symptoms of some duration.

6. *Dementia with Paralysis.*—Dementia with paralysis is often confounded with "general paralysis," and all the more readily as in the former speech may be lamed or stuttering, and the gait impaired, the pupils unequal, and perhaps sluggish. Here, a more or less localized, usually persistent, and purely *paralytic* affection of the muscles is in contrast with the general progressive incoordination of movement found in general paralysis, which, later on, is accompanied by a torpor and paresis of movement as well.

The history of the case also aids in the differential diagnosis. A person in health is stricken with paralysis, often more or less complete, and local, and then gradually grows demented, and, perhaps, weakly emotional. This is not the course of general paralysis. The condition is slowly progressive or stationary, rarely partaking of the special attacks, the crises and fluctuations, so common in general paralysis. In dementia with paralysis, also, there is often early contraction and pain in the paralyzed members, while the dysphasia (if any) is paralytic, and the

* Brown-Séquard, "Archives de Physiologie," 1868, T. i. p. 157;—*Journal de la Physiologie*, etc. vol. i. 1858, p. 472.

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mouth often persistently drawn awry. In it, also, the effects of apoplectic, or apoplectiform, seizures are more decisive and persistent than in general paralysis. The usually greater age of these dements may also assist us in forming a conclusion.

Again, it is only general paralysis *without* ambitious delirium or expansive feeling that is simulated by the condition in question. As compared with the dementia sometimes coming on primarily and exclusively in general paralysis, the dementia following apoplexy is discriminated mainly by the fact that the latter comes on after the truly paralytic symptoms have been established, and often in succession to an apoplectic comatose attack, and then sometimes progresses more rapidly than in this clinical form of general paralysis, and wants its characteristic variety of symptoms (Krafft-Ebing), while the loss of memory may be circumscribed, relating only to a certain order of facts or events; the disorder of consciousness may be less marked than in general paralysis, and motiveless weeping and laughter,—transitory, and frequently recurring,—often separate it from the clinical form of general paralysis now in question. On the other hand, even in the course of the dementia form of general paralysis one may at times be surprised at the unwonted evincement of exalted, or of hypochondriacal delusions of the kind usual in the more classical forms of the disease.

7. *Senile Dementia.*—As Dr. Blandford* says, “senile dementia may be characterized by loss of memory, extravagant and indecent conduct, and delusions. There will, however, be an absence of the specific delusions and the maniacal condition; neither shall we find the inequality of pupils, the stutter, nor stumbling gait. In fact the failing mind in senile dementia is manifested usually long before any symptoms of bodily paralysis, and general paralysis is rare at the age of sixty, senile dementia seldom beginning so soon.”

8. *Senile Dementia with Paralysis.*—But senile dementia with paralysis will simulate some clinical forms of general paralysis more closely. Here, as Krafft-Ebing† indicated, if there is in each a maniacal attack at the beginning, it takes place, in senile dementia, with an absence of the excessive impulse to movement and tumultuous grand delusions of general paralysis; there is instead merely a childish activity and babbling restlessness; while if both begin with progressive weakening of the intelligence, there is in *dementia senilis* less of that disturbance of the consciousness which is shown by mistakes as to persons,

* “Insanity and its Treatment,” London, 1871, p. 279.

† “Allgemeine Zeitschrift für Psychiatrie,” 1866.

times, or places, and more of early failure of memory, than in general paralysis of the kind simulated by it. Again, in *general paralysis* the motor affections are early, general, somewhat changeable, oscillate in their course, and are primarily of the nature of incoordination; while in *dementia senilis* they are usually local, are true palsies, are simply progressive, stationary or slowly improving, and often are accompanied with local contractions, even at an early date.

The disorder of speech in the two affections necessarily partakes of these relative differences: that of senile dementia complicated with paralysis is the imperfect speech of true paralysis, secondary and later in appearance, and the mouth is often persistently awry from partial facial palsy, whereas in general paralysis the imperfection is primarily ataxic, and secondarily parietic, and only finally, if ever, in part paralytic, and is accompanied by tremulous twitching and spasmodic action about the lips, tongue, and face rarely seen in *dementia senilis*. The apoplecticiform attacks, if present, usually leave marked traces behind them in senile dementia with paralytic complication, but their effects are more usually transient in general paralytics. Sometimes grinding of the teeth and epileptiform seizures aid in distinguishing general paralysis from senile dementia.

Sometimes other obvious differences (Krafft-Ebing) betray themselves in the age and sex of the patient, in the exciting causes; and in the shorter duration, less impaired special senses, and ocular peculiarities, of general paralysis.

Doubt only arises in those cases of general paralysis in which dementia predominates throughout; or, again, those in which there is a chronic state of agitation restlessness and incoherence; or, rarely, those with depression coexistent with a shattered intellect.

Since the above was written I notice that Voisin* would extend the domain of general paralysis at the expense of that of senile dementia, claiming that some usually classed under the latter heading should be transferred to that of general paralysis. He also describes a senile form of general paralysis. Nevertheless, judging by mortality-statistics and what one reads and hears, it would seem, rather, that already there is a regrettable tendency in this direction, with the result of thrusting into the domain of general paralysis a host of various senile cases alien to it.

9. *Locomotor Ataxy*.—Dr. John Hitchman † mentions the close similarity sometimes observed between this and general paralysis.

* *Op. cit.*, p. 276, *et seq.*

† "Clinical Observations on the Diagnosis of General Paralysis." *British Medical Journal*, 1871, vol. ii.—Reprint, p. 19.

At their onset the motor symptoms of general paralysis are of the nature of incoordination, and when the gait is affected very early in the disease, and the posterior columns of the spinal cord are implicated, the gait of the general paralytic is somewhat like that of a subject of locomotor ataxy. It is no easy task to distinguish such a case from locomotor ataxy accompanied, as the latter may be, with certain mental symptoms. The absence or mildness of the fulgurant pains, of the early and obvious anæsthesiæ, and of the ocular troubles, of locomotor ataxy, as well as of its not infrequent *early* urinary incontinence, seminal emissions, and engirdling sensations, will help to guide the diagnosis. So also will the presence of the affection of speech of general paralysis which, like its twitching of the lips and face, during articulate utterance, is scarcely simulated in locomotor ataxy. There is, also, the proportional relationship between (1) the degree of disorderly and unsafe locomotion, and (2) the actual failure of muscular power;—the former bearing a much higher comparative ratio to the latter in locomotor ataxy than in general paralysis. For in the motor sphere of neither is ataxia always everything.

In a case of doubt the test of closing the eyes and watching its effect on equilibration and on locomotion should be employed, and the presence or absence of patellar tendon-reflex, or the "knee-phenomenon," should be verified. First observed in the healthy subject by Westphal in 1871, patellar tendon-reflex was found by him to be absent in locomotor ataxy, an observation confirmed by Prof. Erb and O. Berger.* On the other hand tendon-reflex can usually, if not always, be elicited throughout the course of general paralysis;† once only have I found it wanting.

In one class of cases it is believed that true general paralysis may begin by a propagation from the lesions of true locomotor ataxy, as argued and exemplified by Achille Foville,‡ while cases possessing the same pathological import have been described§ by Westphal, Magnan, Motet, Dally, Falret, and Ph. Rey,|| and, perhaps, by H. Hoffman,¶ and by Horn** in 1833.

* *Berliner Klinische Wochenschrift*, Jan. 7, 1878. *London Medical Record*, March 15, 1878. T. Buzzard, *The Lancet*, July 27, 1878, p. 111, and Aug. 10, 1878, p. 175. W. R. Gowers, Royal Med. and Chir. Soc. of London, Meeting of Jan. 28, 1879.

† See also Muhr, "Psychiatrisches Centralblatt." Abstract, *Journ. Mental Science*, Jan. 1879, p. 680.

‡ *Annales Médico-Psychologiques*, Jan. 1873.

§ Méd.-Psych. Soc., Paris.—*Ann. Méd.-Psych.*, Sept. 1872, p. 260.

|| *Ann. Méd.-Psych.*, Sept. 1875, p. 161 (3 cases).

¶ "Zeitschrift für Psychiatrie," xiii. Band, p. 207.

** Locomotor ataxy of nine years' standing followed by general paralysis (?).

Of other kinds than those last mentioned were the cases reported by Mr. J. W. Plaxton,* and apparently some of Dr. P. Nicol's† cases—cases of locomotor ataxy with intercurrent exalted delusions or some buoyancy or optimism passing, or tending to pass, entirely away.

Of other kinds also were certain cases by M. Baillarger, and Herr Westphal.

Thus Baillarger‡ observed general paralysis appear in the first or cephalic period (Duchenne) of locomotor ataxy. According to whom sometimes the former increases and the latter is arrested, sometimes the former disappears and the latter worsens, and in other cases they advance in an equal and parallel manner.

Some years ago Westphal§ described several cases from which he concluded that in a late stage of grey degeneration of the posterior columns of the spinal cord mental disease sometimes supervened; which, by its delirium, paralytic symptoms, intercurrent apoplecticiform, and, occasionally, convulsive, seizures, bore a likeness to general paralysis. Yet in these cases he had not found the same morbid process and change in the brain as in the cord, nor any meningeal adhesion to the cortex, nor, indeed, much worthy of mention, except a certain amount of *hydrocephalus internus*, or central softening, and cortical pallor. Amongst the clinical characteristics separating these cases from general paralysis were the absence of impairment of articulation, and the presence of staggering when the eyes were shut.

In his work on locomotor ataxy, P. Topinard admits a cerebral form of the disease, in which various mental symptoms appear;—also that in a rare form of general paralysis the symptoms of locomotor ataxy follow on a propagation of disease from the brain to the cord.

Steinthal|| had long ago noticed the gaiety and indifference in some subjects of locomotor ataxy.

The ataxic affection with psychic elements described by Kirn¶ of Illenau, also was independent of general paralysis.

It is evident that the relationships between general paralysis and locomotor ataxy remain to be worked out.

10. *Paralysis Agitans*.—A symptomatic paralysis agitans occurring in the course of general paralysis may superinduce

* *Journal of Mental Science*, July, 1878, p. 274.

† West Riding Asylum Reports, vol. i. p. 171.

‡ *Annales Médico-Psych.*, 1862, p. 1.

§ "Allgemeine Zeitschrift für Psychiatrie und Psychisch-Gerichtliche Medicin," Band xx. 1 Heft, p. 1. "Tabes dorsalis (Graue degeneration der Hinterstränge) und paralysis univers. progressiva."

|| Hufeland's Journal, 1844. Quoted by Rey.

¶ "Allgemeine Zeitschrift für Psychiatrie," 1868.

In the initial stage, and even at a later period, the apoplectic-form seizures of general paralysis cannot at the moment be well distinguished from ordinary sudden cerebral congestion. A short period, however, usually suffices for the congestive symptoms to pass away, leaving behind them some ataxy or mental phenomena indicating general paralysis.

From the stupor or coma of actual cerebral hæmorrhage the apoplectic-form seizures of general paralysis may be separated by the symptoms being usually of less gravity, and by the difference in the modifications undergone by the temperature of the body under the two circumstances. In the hæmorrhagic case the temperature falls after the attack, and generally remains below the normal for some time, perhaps twenty-four hours, afterwards rising more or less above the normal, and subsequently pursuing a course which varies very greatly according to the degree of the gravity and fatality of the hæmorrhagic lesion. In the apoplectic-form attacks of general paralysis, on the other hand, the temperature often rises somewhat just before the attack, is high during it, remains high immediately after it, and only subsides with the subsidence of its attendant mental phenomena, or somewhat earlier than do these. Any accompanying paralysis, also, is of shorter duration and is less strictly limited in extent and distribution than that often attending hæmorrhagic effusion.

Yet meningeal and other hæmorrhages may, and do, occur in general paralysis, and produce their usual symptoms, modified by the already existing disease of brain.

13. *Chronic Generalized Palsy.*—The physician may be called to a case of which there is no reliable history, in which the limbs are deprived of voluntary movement, the tongue is inert, and speech lost, and in which the intelligence is so far impaired as to prevent information being obtained from the patient in other ways. This condition, as Calmeil long ago mentioned, may arise from old double cerebral hæmorrhage, or from a general compression of the encephalon of variable origin, as well as from advanced general paralysis, especially if in a “state of complication,” so called. Bucknill* proposed to found the diagnosis of such a condition, when due to the former causes and not to advanced general paralysis, upon “the muscular firmness and power of expression retained by the features compared with the profound palsy of the limbs, and upon the susceptibility of the limbs to excito-motory action,” which latter, on the contrary, was greatly lost in general paralysis.

* “A Manual of Psychological Medicine,” London, 3rd edition, p. 460.

Grinding of the teeth, bending forward of the head, corrugation of the forehead, and some remains of ataxic fibrillary twitching in its accustomed seats would aid here in confirming the existence of general paralysis.

14. *Acute, more or less Generalized, Palsy.*—No doubt could arise as between this and the early and middle periods of general paralysis. From the prostration and, frequently, paralysis of the ultimate period, a suddenly produced generalized palsy may be separated by the history of the cases, and, perhaps, by the completeness of the palsy. A paralysis of this kind may arise from sudden extensive double cerebral hæmorrhage, or softening, or from similar lesions holding median, or symmetrical positions in certain basal portions of the encephalon. With the last-named, however, are special symptoms of bulbar, or of mesocephalic, origin which would assist in the diagnosis.

15. *Generalized or Diffuse Paralysis after Acute Affections.**—Huxham, in his "Essay on Fevers," Frederic Hoffman, and Macario have been among the pioneers on this subject. Beau, Sée, Gubler, Maingault, Trousseau, Westphal, and J. Rose Cormack have assisted in the work; also many others, as Faure, Pidoux, Leudet, Péry. M. Adolphe Gubler.† especially, showed how various were the relations of paralysis with acute disease, and how different the pathological import of each relation. Also, he showed how the *consecutive* paralysis might follow a great variety of acute affections, as pneumonia, erysipelas, cholera, dysentery, typhoid, typhus, and the exanthematous fevers, acute angina, diphtheria, a kind of erythema nodosum, miliary roseola, purpura, febrile urticaria, guttural herpes, and others.

Consisting as these consecutive paralyses do of two varieties,—the localized and the diffuse—it must be borne in mind that it is the diffuse or generalized variety with which alone we are now concerned.

This generalized paralysis, consecutive to grave fevers, etc., must be distinguished from "general paralysis" by the history of the case, often by the more frequent and obvious *preceding* anæsthesia, analgesia, numbness, pricking and arthritic pains, and by the circumstance that it often begins in the velum palati, is not

* *Bull. de Therapeutique*, Dec. 1850.—G. Sée, *L'Union Médicale*, 2nd Sér. T. viii. Nov. 8, 1860, p. 257.—C. Westphal, Abstract, *Journ. Mental Science*, July, 1872.—A. Foville, *Ann. Méd.-Psych.*, Jan. 1873.—J. Rose Cormack, Series of papers, *British Medical Journal*, vol. ii. 1874; vol. i. 1875.

† A. Gubler, "Archives Générales de Médecine," 1860, T. i. pp. 257, 402, 534, 693; 1860, T. ii. pp. 187, 718; 1861, T. i. p. 301.

primarily an ataxy, almost always undergoes recovery in the space of a few weeks, and is rarely accompanied by intellectual trouble. Yet intellectual weakness, also, is sometimes observed, as in certain of the instances mentioned by Westphal* and A. Foville in relation to variola and typhus.

Or, again, after acute febrile and other acute maladies there may be many of the physical signs of general paralysis, and even mental excitement or ideas of grandeur.† This is distinguished from general paralysis by the incompleteness of the picture, and the rapid recovery, although it has been suggested that true general paralysis may occasionally thus take origin, as, for example, after cholera or diphtheria. Insanity after these acute affections has been attributed to a reflex effect on the brain from the periphery. J. Christian‡ reports a case simulating general paralysis and following enteric fever, and a similar one by Max Simon.§

Should it be diphtheritic (and even in some other cases) the paralysis is apt to extend from the velum palati to the pharynx, thence to the lower limbs, then sight and hearing become affected, then the upper limbs, and finally the trunk and respiratory muscles, while the premonitory signs mentioned above are often present.

S. G. Webber|| of Boston, U.S.A., says, "Dr. Mühsam gives three differences between the paralysis following diphtheria and that found after other acute diseases, as cholera, typhoid, and variola. 1st. In the latter, paralysis is found only after very severe cases; diphtheritic paralysis, however, after very light and locally circumscribed cases. 2nd. In diphtheritic paralysis often a longer time elapses between the primary disease and the paralysis, while in the other affections it follows closely upon the acute disease. 3rd. In diphtheria a certain definite group of muscles is attacked in a regular succession; while after the others the most diverse groups of muscles may be attacked.

"Of these differences I think the first does not exist, certainly not to any extent. . . . The other two distinctions are well made, however."

The clinical aspect alone is of interest here, and it is

* *Journ. Mental Science*, July, 1872 (Abs.).—*Ann. Méd.-Psych.*, Jan. 1873.

† Delasiauve, Baillarger, Christian.

‡ "Archives Générales de Médecine," 1873, T. ii. pp. 257 and 421.

§ *Loc. cit.*, p. 269.

|| "Transactions of the American Neurological Association," 1877, vol. ii. p. 152.

unnecessary to refer to the various views and observations as to the pathological anatomy and pathogenesis of the paralyzes after acute affections found in the contributions of Gubler, Charcot, Vulpian, Buhl, Leyden, Oertel, Bernhardt, Nothnagel, Westphal, Voisin, Ebstein, Pierret, Déjerine, Brown-Séquard, Letzerich, F. Magne, and others.

16. *Acute Ascending Paralysis*.—The history and rapidity of the case, and comparative or complete absence of mental symptoms, the paralysis instead of incoordination with paresis, and the special lines of invasion of the musculature, also separate from general paralysis, another class of cases which has sometimes been called acute ascending paralysis. This, however, like the last group, may follow acute febrile affections. It usually pursues a certain march from the toes and feet to the back of the thighs and pelvis, thence successively to the front and inner parts of the thighs, the fingers, hands, arms, scapular region, biceps, trunk, respiratory muscles, tongue, etc.*

17. *Plumbism*.—Cases not unlike certain conditions of general paralysis have been observed in the course of lead-palsy, as in cases by Desvougues and Delasiauve, and as in a case of acute diffuse paralysis recently reported by T. S. Dowse.† Here the history of the case, the complete cutaneous and muscular anæsthesia from the level of the *alæ nasi* to the soles of the feet, the total failure of muscular response to the induced electrical current, the trismus, attack of tetanic spasms, and saturnine gum-line, serve to separate this case from the final stage of general paralysis.

The brain and spinal cord contained lead. The spinal cord was congested, its vessels were dilated, it contained colloid bodies, and some grey degeneration which became more and more manifest as the examination proceeded upwards, and most of all in the medulla oblongata, as abundant miliary sclerosis and scattered colloid bodies.

18. *Chronic Softening of the Brain (local)*.—Chronic softening must be diagnosed from general paralysis by the history of the case, the localization of the motor symptoms, the absence of the characteristic early irregularity and incoordination of move-

* *Gazette des Hôpitaux*, Sept. 10, 1859, p. 421; Sept. 17, 1859, p. 433.—Alf. Liégard, *Ibid.* Dec. 3, 1859, p. 562.—O'Landry, *Gaz. Hebdomadaire de Médecine et de Chirurgie*, July 29, 1859, p. 472, and Aug. 5, p. 486; and cases by Ollivier (d'Angers) and Sandras.—Geo. Harley, *The Lancet*, vol. ii. 1868, p. 451.—Some of Gubler's cases (*suprà*).

† "Transactions of the Clinical Society of London," vol. viii. 1875, p. 124.

ment seen in general paralysis, and by the absence, also, of mental symptoms of the kind more usual in general paralysis, as well as, often, of its more special spasmodic phenomena.

19. *Tremors of the Aged* when observed in the insane must not be confounded with those of general paralysis. They do not produce the peculiar speech of the latter, are not affected by its complications, nor accompanied by the same mental phenomena.

20. *Defective Speech*.—Nor does the least difficult case arise when one becomes insane who for long has suffered from an impediment in speech. And especially is the diagnosis difficult here if expansive feeling or idea displays itself. The absence of that state of pupils, tongue, gait, writing, and sensory function, both special and general, usual in general paralysis, and of the special characters of its delirium, serve to distinguish these cases of speech-defect with mental alienation—but not so readily, by any means, as might be surmised.

CHAPTER VI.

CAUSES OF GENERAL PARALYSIS.

A. *Predisposing Causes.*

Of these the principal may be considered under the heads of sex, age, temperament, mental activity, character, heredity, condition in life, occupation, mental causes, previous mental disorder, cessation of discharges, climate, and habitat.

(1.) *Sex*.—The relative proportion in which general paralysis attacks the two sexes is usually that of between four and five males to one female:* some have found the proportion less unequal; others, as Ramon,† have placed it as high as eight males to one female. Speaking in 1826 of the inmates of Charenton A. L. J. Bayle‡ reported a little over *one fifth* of the *males*, and *one-twenty-eighth* of the *females*, as being general para-

* Thirty-first Report (1877) of the Commissioners in Lunacy for England, Table xviii.—R. Boyd, *Journal of Mental Science*, April, 1871, p. 17.—Burman, West Riding Reports, vol. i. p. 129.

† Males more than one-fifth (60 in 286); females one-thirty-ninth (5 in 195): or, about 8 males to 1 female.

‡ “*Traité des Maladies du Cerveau et de ses Membranes*,” 1826, Introduction, p. xxvii.

lytics, or a proportion of almost six males to one female. Now, at the very same period, and drawing his statistics from the same asylum, L. F. Calmeil* only spoke of *one-fifteenth* of the male cases, and *one-fiftieth* of the female, as suffering from that affection. But by 1859 he† gave the proportion of male general paralytics to the total male admissions into the asylums of French cities as from one-fourth to one-third. This discrepancy between the statistics of Calmeil in 1826 and 1859 cannot have been due entirely to a greater prevalence of general paralysis at the latter date. It seems to show that in 1826 Bayle had more accurately recognized the disease than had Calmeil at the same period, for, as we have just seen, at that time, of the two, the statistics of Bayle were much more in accord with the modern statistics of the disease.

At an intermediate date Esquirol stated that during a certain period more than one-fourth of the male admissions (95 in 366) into Charenton, and one-eighteenth of the female (14 in 253), were those of "paralytics."

H. Schüle (Ziemssen's Cyc.) admits a proportion of female to male paralytics as two to five; and, like others, he notes the greater tendency to primary mental weakness and the gentler form of grandiose ideas in the females.

Of the patients admitted during a quarter of a century into the Devon County Asylum about 14 per cent. of the males and about 3 per cent. of the females were general paralytics. (Burman, *loc. cit.*)

From Table xviii., pp. 40-1, of the Thirty-first Report of the Commissioners in Lunacy (England) it appears that of the admissions into lunatic asylums in England and Wales during the year 1876-987 of the 6,973 male admissions, and 234 of the 7,179 female admissions, were returned as general paralytics:—or, in other words, 14.1 *per cent.* of the total male admissions, and 3.2 *per cent.* of the total female admissions, were general paralytics.

From this, and from the succeeding report,‡ Dr. T. Algernon Chapman§ has constructed the following table:—

"TABLE G.

"Number of General Paralytics that would occur at each age and condition as to marriage among the number of males living, that among females yielding one General Paralytic.

* "De la Paralyse Considérée chez les Aliénés," 1826, pp. 370, 371.

† "Traité des Maladies Inflammatoires du Cerveau," 1859, T. i. p. 270.

‡ Thirty-second Report of Commissioners in Lunacy (England), 1878.

§ *Journal of Mental Science*, April, 1879, p. 37.

Predisposing Causes of General Paralysis—Sex. 91

“Ages.

| Condition as to Marriage. Males. | Under 20 | 20 | 30 | 40 | 50 | 60 | 70 | |
|-------------------------------------|----------|-----|-----|-----|------|-----|-----|---|
| Single | 1 | 4 | 5·3 | 5·8 | 12·8 | 4·3 | ... | } Each to one female general paralytic. |
| Married..... | ... | 9 | 4·8 | 3·6 | 6·0 | 7·8 | ... | |
| Widowed | ... | ... | 6·0 | 3·0 | 6·4 | 7·9 | 6·0 | |
| Total (Males)... | 1 | 4·7 | 4·9 | 3·7 | 8·0 | 7·8 | 3·0 | |

“This shows that amongst the single the proportionately greater frequency of general paralysis amongst males increases with age, whilst among the married and widowed it is at a minimum between forty and fifty, the age of greatest prevalence of general paralysis in both sexes.”

Several of the earlier observers attributed this disproportion between the numbers of the two sexes affected with general paralysis to a prophylactic influence of the menstrual discharge in women: R. Daveau* asserted this long ago; Lunier said the disproportion between the numbers of the sexes affected becomes less after the age of forty-five owing to the occurrence of the menopause about that period and a consequent increase in the number of female cases; and J. A. G. Doutrebent† and others took much the same view. But it is futile to strain after an explanation; for the statement itself is only partially true. As we have just seen, in this country it is true only of widowed and married general paralytics; the sexual disproportion augmenting on the other hand among single general paralytics at the age in question; while, whatever the condition as to marriage, the *relative* proportion of female general paralytics is lower after the age of fifty than at any time before it; or, in other words, the sexual disproportion is then higher.

Probably the cause of the disproportion mainly lies in the greater moral shocks and mental strain to which the male is subjected, as well as to the greater frequency with which he indulges in excess, especially alcoholic excess.

The main conclusions of a recent inquiry into the subject of general paralysis as found in women, by Dr. Jung,‡ are that general paralysis is increasing in women of the lower class, in them is a disease of the climacteric period, occurs later than with men, seldom is rapid in its course; is favoured by heredity, by

* “Dissertation sur la Paralyse Générale observée à Charenton.” Thèse, 1830.

† “Recherches sur la Paralyse Générale Progressive.” Thèse, 1870.

‡ “Allgemeine Zeitschrift für Psychiatrie,” xxxv. Band, 6 Heft, p. 625.

congenital or acquired weakness of nervous system, or vaso-motor disturbances. The women affected were barren, or had one child only, or the children were still-born, or died when young.

It is difficult to say how far the apparent increase of general paralysis among women of late years is due to a former defective recognition of it, and faulty diagnosis, owing to the less salient features and less dramatic course of general paralysis as it occurs in women, than as in men.

2. *Age*.—General paralysis may occur during the period between the ages of twenty-five and sixty, very rarely before or after. Guislain,* however, saw it occur at the age of seventeen, and Clouston† observed a case at the age of sixteen. Several cases related by Morison‡ appear to have been general paralysis, and of these one was aged nineteen. As for general paralysis in the aged, most of those writ down as such are, in truth, examples of ordinary paralysis, or of feebleness, associated with senile, or other, mental defect or disorder.

In civil life it occurs for the most part between the ages of thirty and fifty-five years. Distributed among the several decades, it is most frequent in that between the ages of forty and fifty,§ even when the statistics include a military element, although it must be confessed that J. T. Austin found it most frequent in the decade from thirty to forty. In the British Army and Navy, however, it is different, and among the soldiers under my care at Grove Hall, and among the sailors at the Royal Naval Asylum at Yarmouth, general paralysis is most common in the decade from thirty to forty years of age. Thirty-three years was the stated *average* age of the soldiers here, at the time when the mental symptoms of general paralysis were first recognized.

This fact is associated with several others which together form an important chapter in the natural history of the disease, and by which it appears that the classes in which general paralysis is most rife are those in which, on the average, it occurs at an earlier age, runs a more severe course, is of shorter duration, and perhaps less often exhibits any remissions or apparent recoveries.

The *average age at death* of 197 male general paralytics at Wakefield, Yorkshire, was 41·7 years; and of forty-six females, 42·2 years.¶ Of 600 male cases reported by Baillarger¶ the

* "Leçons Orales sur les Phrénopathies," Gand, 1852.

† *Journal of Mental Science*, Oct. 1877.

‡ "Cases of Mental Disease," by Alex. Morison, 1828, p. 61.

§ Bayle, Calmeil, Baillarger, Boyd, Burman.

¶ Crochley Clapham. West Riding Asylum Reports, vol. vi. p. 21.

¶ *Gazette des Hôpitaux*, July 9, 1846, p. 317, No. 80, T. viii. 2^o Sér.

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average age on admission to a Parisian asylum was forty-two years;—of 400 females, forty-one years. In the Devon Asylum, of 276 male cases of general paralysis the *average age on admission* to asylum was forty-two years, and of sixty-five females, thirty-eight and a half years (Burman, *loc. cit.* p. 132). To the Parisian statistics, and to the Devonian, a year or more must be added to bring the cases up to the *average age at death* and thus render them comparable with those, just cited, in Yorkshire. From which comparison it may be learned that general paralysis occurs at an earlier average period of life in the last-named than in either the Parisian or Devonian cases, except only for its comparatively early occurrence in Devonian women.

In the most recent statistics* it is found that of the general paralytics admitted, into the Asylums of England and Wales in 1876 the number then actually between the ages thirty to forty was larger than that of those between the ages forty to fifty (as 472 to 415); but that the proportion per cent. of the general paralytics admitted, to the total number of patients, of the same decade-age, admitted to the same asylums during the same year, was greater in the decade forty to fifty than in that from thirty to forty (as 14·4 per cent. to 13·5 per cent.).

In reading the works of the earlier observers one is led to think that formerly general paralysis occurred rather later in life on the average than is nowadays the case; of 182 cases Bayle† had seen only four under the age of thirty, and Calmeil,‡ referring to a somewhat smaller number of cases says he had seen only two under thirty-two years of age. Certainly, there is no rarity of cases under the age of thirty, still more of thirty-two, nowadays. Nor was there even twenty or thirty years ago. Austin's§ statistics show that those under the age of thirty comprised seven per cent. of the cases; Baillarger|| found that in Paris they amounted to one-fifteenth, or about the same; at the Devon Asylum (1845–1870) they comprised one-twelfth, whilst in the soldiers under my care during the past eight years *nearly one-third* were under that age when attacked.

The above impression, if correct, would speak ill for the vitality of the peoples of the West of Europe, as far at least as the

* Thirty-second Report (1878) of Commissioners in Lunacy (England), p. 33, Tables xvi. and xvii.

† *Op. cit.*, 1826.

‡ *Op. cit.*, 1826.

§ *Op. cit.*, 1859.

|| *Gazette des Hôpitaux, loc. cit.*, 1846.

disease may be deemed analogous to a prodigal wasting of vital power, and premature senility; the early attainment of old age in the individual members of a race being the forerunner and prophet of its imminent decay.

3. *Temperament.*—The sanguine temperament has usually been viewed as predisposing to general paralysis. This, perhaps, is true only of one class of the cases; certainly the sanguine temperament is very often absent.

4. *Mental Activity.*—As to the state of intelligence, the same rôle has been attributed to an energetic mental life with ardent imagination, and this no doubt is an active predisponent.

Character or Disposition.—The character or disposition of those who become general paralytic has often by nature been fiery, choleric, intolerant of opposition; or, again, proud, haughty, selfish, ambitious; or, on the other hand, dounce, gentle, and evidencing much *bonhomie*.

5. *Heredity.*—Heredity is a factor in a proportion of cases, Calmeil* says in about one-third. The apparent direct hereditary predisposition in these cases may be either to insanity, or, secondly, to some other and ordinary nervous† disease, and the occurrence of the latter in the family history has been accentuated by several writers. Thus, according to Lunier‡ it is rather that the ancestors of general paralytics have been apoplectic, paralytic, epileptic or demented than insane, and others§ assert that the heredity of general paralysis is very indirect, and consists in the inheritance of a tendency to cerebral congestion, which congestion produces the predisposition to general paralysis; those born of sanguine parents with apoplectic tendencies, and who themselves are subject to cerebral congestion, are predisposed to general paralysis. And Verga|| asserts that "the hereditary affinities of general paralysis are not with ordinary insanity, but with paralysis, apoplexy, and other brain diseases."

Of forty-nine cases at an English asylum hereditary predisposition (to insanity) was found in eight, or about 16 per cent. On the other hand, of 109 cases in New York,¶ in thirty-nine (or about 36 per cent.) was found a history of insanity in one or other

* "Traité," etc., 1859, T. i. p. 272.

† Using the term in wide sense as including both neural and adneurial affections.

‡ *Ann. Méd.-Psych.*, 1849.

§ Doutrebente, Thèse, 1870.

|| Abstract, *Journal of Mental Science*, April, 1873, p. 158.

¶ A. E. Macdonald, *American Journal of Insanity*, April, 1877, p. 451.

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of the family branches—in thirty a history of other nervous diseases; and in twenty-two of intemperance in the parents. Yet to heredity is assigned a part in only 8·4 *per cent.* of the cases of general paralysis tabulated in the English Lunacy Blue Book for 1878. (32nd Report, p. 34.)

6. *Condition as to Marriage.*—General paralysis occurs more frequently among the married than the single. In a New York Asylum while 33½ *per cent.* of other cases of insanity were among the married, as many as 81 *per cent.* of the general paralytics were married (A. E. Macdonald). Of 330 cases in an English County Asylum, 215 were married, eighty-six single, and twenty-nine widowed; and it was noted that the proportion of *single* persons was much higher among the females than among the males. (Burman, *loc. cit.* p. 133.) Austin (*Op. cit.*) observed the same high relative proportions at Bethnal House among *single women*. His tables refer to eighty-five married, thirty-eight single, and twenty-four widowed general paralytics. Nevertheless, larger and more recent statistics refute these, showing that in this country at large the proportion of *single* persons among the female general paralytics is equal to, or less than, that of single persons among the male. In the English Lunacy Blue Book for 1878 (32nd Report, Table xvi. pp. 32–3) it appears that of 1,221 general paralytics admitted into asylums in England and Wales in the year 1876—213 were *single*, 890 *married*, 103 *widowed*, and in fifteen the condition as to marriage was *unknown*. But here the proportion of single to married female general paralytics, is precisely the same as that of the single to married male general paralytics, namely, about 24 *per cent.* (1 to 4). On the other hand, among the *total* admissions of insane and idiots to the same asylums during the same year nearly as many were single as married, the numbers being 6,120 single, 6,340 married, 1,567 widowed, and 127 unknown. Of these 3·4 *per cent.* of the single, 14 *per cent.* of the married, 6·5 *per cent.* of the widowed, and 11·8 *per cent.* of the “unknown,” were general paralytics. (*Ibid.* Table xviii.)

Thus there is an overwhelming majority of married general paralytics.

The time of life at which general paralysis is most rife partly accounts for this fact, inasmuch as it is also a time of life at which the majority of persons in this country are married. How then to arrive at a just comparison? The best method is to find the ratio of general paralytics in the different conditions of life as to marriage, and in the several decades, to the entire population of the same conditions of life and ages. This has been

worked out by Dr. T. Algernon Chapman* from the tables of the Commissioners in Lunacy. First he shows that in the general population between the ages of thirty and sixty the single are more liable to *insanity* than the married in the proportion of 2·83 to 1,—yet that, calculated in the same way, there is only a very slight difference in the frequency of *general paralysis* according to the condition as to marriage. This he attributes to the comparative rarity of congenital defect in general paralytics—defects on account of which so large a percentage of the population remains single:—single, because of the same factor as renders them liable to insanity; *not*, liable to insanity *because* single. Here is his table:—

“TABLE H.

“Ratio, per 100,000, of the General Paralytics, between thirty and sixty years of age, admitted into asylums during 1876 to the whole population of those ages, at the time of the census of 1871, arranged according to condition as to marriage:—

| | Males. | Females. | Total. |
|-------------------|--------|----------|--------|
| Single | 30·4 | 5·4 | 16·5 |
| Married | 24·6 | 5·7 | 15·3 |
| Widowed | 29·1 | 7·0 | 15·4 |
| Total | 26·2 | 5·8 | 15·6 ” |

7. *Occupation.*—Military and naval life; occupations exposing the workers to great heat and sweating, or to alternate heat and cold draughts; prostitution; all favour the production of general paralysis. So do those which entail emotional strain, constant worry and irritation, or intellectual overwork, as the occupations of professional and literary men.

In the Lunacy Blue Book for 1877 the proportion of “persons engaged in the defence of the country” (*males*, of course) to the total *male* population of England and Wales is stated to be as 6 to 1,000 (Table xiii.). The proportion of *lunatics* to *persons* in the same order of persons engaged in the defence of the country is stated as being 31·6 per 10,000, or ·316 per cent., a proportion of lunatics much higher than in any other order of persons (*males*); the proportion in all the “orders” together being 6·3 (*males*), or ·063 per cent.

Yet according to the eighteenth table of the same Report, of the

* *Journal of Mental Science*, April, 1879, p. 37.

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military and marines who became insane 13·6 per cent. were general paralytics, whereas 14·1 per cent. of the *total* males of all orders were general paralytics. Thus, while mental alienation at large is far more rife in the army and navy than in any other order of the male population, yet a slightly smaller percentage of the military insane are general paralytics than of the male insane of the country generally. The recent "short service system" and comparative youth of our soldiery is no doubt the explanation of this. But many included in these returns have no doubt been so long retired from, or so short a time in, the military and naval services, as that their cases have little bearing on the genesis of general paralysis in the army and navy.

Of the 508 soldiers *newly admitted* under my care at Grove Hall Asylum, London, up to July 1879, ninety four were general paralytics, or a proportion of 18·5 per cent. These are exclusive of the general paralytics found among the insane military inmates of the asylum when I took office, and, of course, exclusive, also, of the general paralytics here belonging to other classes of society.

The view of Dr. Sankey that general paralysis is more rife in males of the lower, than of the upper, classes requires confirmation, and is opposed to the experience of Esquirol who found much more general paralysis among the males of Charenton than among those of the Bicêtre; of the two the former, at that epoch at least, having had greater pecuniary resources, readier means of indulging their passions, and higher intellectual activity. It is opposed, moreover, to the general conclusions to be drawn from the eighteenth table of the Lunacy Blue Book for 1877, containing the percentages of general paralytics, among those insane and admitted to asylums, in various "orders of persons." Taking of course the male column only, in this relation, the percentages of general paralytics, among the insane, are these:—

Orders of persons:—Engaged in government of country 24·1 per cent.,—in its defence 13·6 per cent.,—in learned professions, literature, art, science, etc. 12·8,—in entertaining, and performing personal offices 14·5,—in commerce 18·6,—in commercial conveyance 21,—in agriculture 10·1,—mechanists 17·5,—textile fabric-workers 14·2,—workers in food, drink, etc. 16·8,—mineral workers 18;—aristocrats, and plutocrats 7·9, and various 3 per cent.

Predisposing Mental Causes.—A life absorbed in ambitious projects, with all its strenuous mental efforts, its long-sustained anxieties, deferred hopes, and straining expectation; or any prolonged and violent, or sudden and frequent, feeling or passion, whether of worry, indignation, rage, or lust; chagrins, forced

erethism of the intellectual faculties, intellectual overwork, especially if sustained by stimulants, exposure to constant sources of annoyance—all these predispose to general paralysis. Its frequency among educated, highly excitable men, as poets, musicians, and literati has been noticed, amongst others, by Griesinger and Calmeil, nor, recollecting the asserted affinities of general paralysis, is it without interest to bear in mind that Alberti long ago described the frequency of apoplexy among the learned.

a. Previous Mental Disorder or Defect.—To a previous attack of insanity Calmeil would attribute an influence predisposing to general paralysis, but the correctness of this view has been impugned by many. In several cases I have known general paralysis come on in those who had recovered from a previous attack of mental disorder—perhaps years before. Köhler observed general paralysis supervene in an imbecile. (Abs., Journ. Ment. Sci., Jan. 1878, p. 605.)

β. Transformation of Simple into "Paralytic Insanity".—One scarcely need refer to the error of many of the earlier writers who, not knowing general paralysis as a separate affection, viewed its physical signs as complications or terminations of ordinary insanity. Nevertheless, general paralysis may appear to supervene on simple insanity of longer or shorter duration, or, as some would phrase it, simple insanity may undergo a transformation into paralytic insanity (general paralysis). Cases of such asserted transformation may be found in comparatively recent works,* but many of them are by no means satisfactory or convincing.

I have, however, observed the physical signs of general paralysis first appear as late as a year and a half, and more than two years, after the onset of frank mental derangement.

Cessation of Discharges.—A suppression of lactation, or a suppression of the menses, seems occasionally to act as a predisponent; so does suppression of hæmorrhoidal flux, and, doubtfully, of discharge from ulcers, or, that of chronic dartrous affections.

Cranial Injuries must also be mentioned here.

Climate and Race.—General paralysis is said to be rare in hot climates, but it is an old observation that this comparative exemption does not apply to the new comers there. It affects Saxon and Celt alike, but Ireland and, to a lesser extent, the

* Calmeil, "Traité des Mal. Infl. du Cerv.," T. i. p. 434, 8th Series (3 cases).—Burlureaux, *op. cit.*, p. 41, *et. seq.* (2 cases).—Voisin, "Traité de la Paralyse Générale des Aliénés," p. 361 (2 cases).

north of Scotland appear to be comparatively free from it.* It is said not to have been recognized in America until 1843, when Dr. Luther Bell first announced its existence there. Now, at least, it is common enough on that continent. As for Canada; Dr. J. Workman† writes: "When I entered the Toronto Asylum in 1853, there was not a single case, as far as I could judge, in the institution, but it was not long before it began to make appearance. . . . in my last ten and a half years, from Jan. 1865 to July 1875, the deaths from (general) paresis amounted to seventy-two." As for the United States:—Of 1,600 patients under treatment, during two and a half recent years, at the New York City Asylum for the insane it is stated‡ that 205 were general paralytics. If reliable, the asylum reports would tend to show that general paralysis is much more rife in the eastern and middle than in the western and southern parts of the United States of America.

Comparing general paralysis in England and America, the proportion, *per centum*, of general paralytics to the *known insane* of all varieties is:—

| | | |
|---------------------------|--------------------------|---------------------------|
| In England, . . . | 14·1 p. c. of the males; | 3·2 p. c. of the females. |
| In the United States, 4·1 | " " | " 4 " " |

And of the deaths among the insane in a number of asylums there were, according to Dr. A. E. Macdonald (*loc. cit.*), from general paralysis:—

| | | |
|----------------------------|--------------------------|----------------------------|
| In Great Britain, . . . | 25·5 p. c. of the males; | 12·5 p. c. of the females. |
| In the United States, 19·4 | " " | " 5·8 " " |

He also avers that statistics show an uniform order of progression in each country.

1. The appearance and recognition of the disease in males.
2. Increased frequency of occurrence in male patients, and appearance in female.
3. Increased frequency of occurrence in both sexes, and in larger proportion than the increase of ordinary forms of insanity, and increase in the proportion of females to males attacked.

Though unproven this, indeed, is what one would expect; but the numerical returns vary extremely with the varying knowledge of the disease enjoyed by different alienists and medical practitioners.

* 18th Annual Report (1876) of General Board of Commissioners in Lunacy for Scotland. *Journal of Mental Science*, Oct. 1875, p. 465, et seq. *Ibid.* April, 1876, p. 82 (I. Ashe).

† *The Canada Lancet*, August, 1878, p. 356.

‡ *American Journal of Insanity*, April, 1877, p. 451.

So that many statistics are utterly misleading as guides to a knowledge of the relative proportions of general paralysis in different countries and in different localities, and are, in part, merely an index of the varying capacity of the medical men to *recognize* the disease. In truth, the above assumed mode of incidence and growth of general paralysis, in a people, is not well supported by the figures quoted in previous sections when speaking of *age* and *sex*. To a large extent the American, British, and French statistics in this particular relation do but limn the lines along which a practical knowledge of the disease grew in the minds of asylum physicians, the more obvious and numerous male cases being recognized first,—the less striking, and less frequent, female cases afterwards.

Urban Life.—Town life, rather than rural, seems to foster general paralysis. In the Scottish Lunacy Blue Book for 1876* are some statistics bearing upon this subject, and indirectly upon the causation of general paralysis by alcoholic excess. According to these, in the *town* districts of Scotland general paralysis yielded an annual death-rate of 1·9 per 100,000 of the population; but only ·8, or less than half, in the insular and mainland rural districts. Or, taking another and subdivided classification, the annual death-rate, per 100,000, from general paralysis was,—in the *principal towns* 2·1, in the *large towns* 1·3, and in the *small towns and rural districts* only ·7. Moreover, “while the death-rate from general paralysis is three times as great in the *principal towns* as in the *small towns and rural districts*, the death-rate from *all* causes is considerably less than twice as great.”

Again, the annual rates of mortality, per 100,000 of general population, in Scotland are:—

| | All causes. | Diseases of Brain and Nerv. Syst. | Delirium Tremens. | General Paralysis. |
|--------------------------------------|----------------|---|----------------------|-----------------------|
| In insular and mainland rural parts, | 1,841 | 21 | 1·2 | ·8 |
| In towns, | 2,565 | 35·6 | 2·6 | 1·9 |

For every 100 deaths from all causes in the insular and mainland rural districts there are 139 deaths in the towns.

“For every such 100 deaths (*i.e.* in the country districts) from nervous diseases there are 170 in the towns. For every 100 deaths from delirium tremens there are 217 in the towns: and the corresponding proportion for general paralysis is 237.”

* Eighteenth Annual Report of the General Board of Commissioners in Lunacy for Scotland.

B. Exciting Causes.

Many have held that these exciting causes act by producing cerebral congestion, upon which they lay peculiar emphasis as being the proximate or efficient cause of general paralysis. So far back as 1822 A. L. J. Bayle had attributed this rôle to all the causes, and in 1830 R. Daveau spoke of cerebral congestion as the great exciting cause;—congestion which may be sudden and apoplectic in its onset, or slow, and evinced by heaviness, dull weight in the head, confusion on awaking, forgetfulness, and vertigo.

Prominent among the exciting causes are alcoholic excesses, sexual excesses, cranial injuries, exposure to extreme solar heat, violent and protracted, generally painful, emotions, especially when associated with excessive intellectual labour and deficient time for sleep and recreation. To these add prolonged play of the passions alone, such as rage, jealousy: thwarted ambition, disappointed and helpless pride, thirst for wealth, anxiety due to actual, to prospective, or to merely anticipated loss of means; or, again, dread of impending danger whether actual, or threatened, or imaginary. As Calmeil (*Traité, &c.*, T. i. p. 271) says, many of those who sink under one or several of causes such as these have been addicted in youth to excess of study, of night-work, or of venery, or have used mercury in excess, or have discontinued their customary habitual venesection.

M. Böens* seems to have little ground for his assertion that religious fanaticism is the most potent of the moral causes of general paralysis.

Suppression of hæmorrhoidal and other fluxes has been alleged as a frequent exciting cause, while the abuse of mercury, of opium, and of tobacco have each been credited with producing the disease, and the same rôle has even been suggested for lead. We have just mentioned the old dictum that the excessive use of tobacco may produce general paralysis; it has recently been re-asserted by Lefebvre. (*Gaz. Hebd.*, Jan. 1874.)

In the causation of general paralysis Dr. Hack Tuke† gives prominence to alcoholic excesses and dissipated habits, combined in many cases with insufficiency of nourishment.

Verga summed up the effective causes as being, in general terms, "abuse of the moral and intellectual powers or of the cerebral functions."

The statistics collected by Burman (*loc. cit.* p. 41-2) show a slight excess in frequency of the moral over the physical causes;

* *Gazette Hebdomadaire*, Jan. 1874, p. 91.

† "A Manual of Psychological Medicine," 3rd edition, p. 334.

Unfortunately these proportions cannot well be compared with the percentages given in Table xiv. p. 32, in the preceding (31st) Report, of the assigned causes of insanity in *all* the patients admitted during 1876 into the asylums of England and Wales. For the table of these percentages is constructed upon a basis different from that of the table in the 32nd Report, relative to the assigned causes of the insanity in the general paralytics admitted during the same year. Thus it comes about that in the table relating to *all* the cases admitted, and as explained in the Report itself, the "totals represent the entire number of instances in which the several causes (either alone or in combination with others) were stated to have produced the mental disorder. The aggregate of these totals (including "Unknown") will, of course, exceed the whole number of patients admitted; the excess is owing to the combinations."—Whereas, the "totals" in the table, in the 32nd Report, relating to the assigned causation of general paralysis only amount, as already stated, to two-thirds of the whole number of general paralytics admitted.

Read on the surface only, these tables would assign to general paralysis, as compared with the total insanity (and idiocy) of the country ;—*Less frequent* moral causes (with the exceptions named below), *less frequent* hereditary and congenital causes ; also the following as *less frequent* causes,—self-abuse, pregnancy, puerperal state, lactation, puberty, and change of life, uterine disorders, fevers, and “other bodily diseases or disorders including old age.”

On the contrary, they would assign the following as *more frequent* causes of general paralysis, relatively to insanity at large :—Intemperance in drink, sexual intemperance, accident or injury, overwork, sunstroke. Also, among the *moral* causes, “adverse circumstances, including business anxieties and pecuniary difficulties ;” and (in the male only) “mental anxiety—‘worry.’”

I think the following efficient causes of over-excitation and exhaustion of the brain frequently bring on general paralysis, and of the last four each is associated in many cases with undue alcoholic stimulation.

I. Alcoholic excess.—II. Excessive and prolonged intellectual labour, with undue emotional tension.—III. Protracted painful emotional strain.—IV. Exhausting heavy physical labour.—V. Sexual excess.

Very similar is the summary of Dr. John Hitchman.* “The most energetic agencies in the production of the disease, in the more opulent classes of society, are, I believe, intense and prolonged mental activity, carried on under emotional excitement ; sexual excesses ; and especially if under either of these circumstances large quantities of wine and alcoholic stimulants be resorted to ; among the working classes heavy and prolonged labour, sustained by large potations of ale or spirits rather than by nutritious food and a due quantity of sleep.”

I would reject the view of Burlureaux† that true general paralysis may be produced by the inflammation occurring around cerebral tumours, or around *foyers* of softening, and gaining the periphery of the brain and the meninges.

We will speak separately of (a) alcoholic excess ; (β) sexual excess ; and (γ) moral causes.

(a.) *Alcoholic Excess*.—Drink is a fertile cause of general paralysis. According to Thomeuf‡ of 350 admissions of insane at Charenton 102 were attributed to drink, and of these 34 were general paralytics. A similar conclusion was previously

* *British Medical Journal*, vol. ii. 1871 ; and Reprint, p. 7.

† *Op. cit.*, p. 76, and *Gazette Hebdom.*, Jan. 16, 1874.

‡ *Gazette des Hôpitaux*, July 19, 1859.

reached by Guislain (*op. cit.*, T. ii. p. 55); and by Comtesse ("Études sur l'Alcôolisme") with regard to 8 per cent. At the New York City Asylum a history of alcoholic intemperance was obtained in 116 out of 155 general paralytics. (Macdonald, *loc. cit.*, p. 467.) Describing alcoholic excesses as producing any form of mental disease, Guislain adds that general paralysis is the form of phrenopathy more particularly engendered thereby. Even here to the alcoholization are usually added the effects of chagrins or of fatiguing intellectual labour. To *sensual* excesses of all kinds (alcoholic, sexual, dietetic, etc.) half the cases of general paralysis are assigned, he says, in etiological records.

In my own cases alcohol, though perhaps rarely acting alone, has appeared to be by far the most frequent and efficacious cause of general paralysis.

Dr. Auguste Voisin (French Méd.-Psych. Assoc., May, 1872), however, declared that general paralysis cannot be produced by alcoholic excesses, and that the anatomical changes were essentially different in cases of the two classes. In alcoholic cases the cerebral lesions were fatty and atheromatous degenerations, dilatation of arteries, sanguineous exudates in the vascular sheaths and in the nervous substance, cerebro-meningeal congestion; œdema; while on the other hand he noticed that hypertrophy of connective tissue and proliferation of nuclei were absent. But in his work published since the above sentence was written he partially recedes from that position, acknowledging alcoholic excess as a predisposing cause. (*Traité, &c.*, 1879, p. 320.)

Nevertheless, in the last Lunacy Blue Book (1878) one-third of the cases in which the causes were assigned for general paralysis were attributed to "intemperance in drink," the numbers being 272 out of 814, or just one-third of the 66·6 per cent. of total admissions, of general paralytics, in which any causation was assigned: alcoholic intemperance, therefore, appearing as by far the most frequent assigned cause of general paralysis.

(β.) *Sexual Excess* (excess of coitus).—J. Guislain is sometimes quoted in support of the view that would assign a peculiar potency to sexual excesses in the production of general paralysis. What Guislain really says is briefly this (*op. cit.*, pp. 55 and 64). Sexual excesses when conjoined with other debauchery, as alcoholic excess, tend to terminate in general paralysis. At the same time there is nearly always a predisposition, or the operation of a moral cause of the disease. We must not then always assign sexual excesses or spermatic emissions as the causes of general paralysis; excessive drink, fear, various reverses and disappointments, can directly bring it on, and intellectual overstrain

may also lead to it. Indeed, he saw a girl, aged seventeen, a pupil at a "normal school," stricken with general paralysis after severe over-study; and elsewhere he mentions dry intellectual work among the causes.

By Sankey (Lectures, &c., 1866, p. 181) it has been deemed "remarkable how many general paralytics had led irregular lives, and especially had been guilty of sexual impropriety of some sort," and he tried to prove the existence of an inverse proportion between the relative degree of subjection of the passions in several classes of persons, and the prevalence in them of general paralysis; and, on the other hand, a direct proportion between the prevalence of general paralysis and of syphilis in the same several classes. Even if true, this does not establish any connection between the syphilis and the general paralysis. Dr. E. Sheppard agrees with the last-named author as to the very prominent part held by sexual excess in the etiology of general paralysis. (Med. Times and Gaz., vol. i. 1873, p. 163.)

At a meeting (Jour. Mental Sci., April, 1873, p. 166) of the Medico-Psychological Association in London an animated discussion occurred on the subject of sexual excess considered as the most fertile cause of general paralysis; this thesis being supported by Dr. Maudsley and others, but being opposed by several members. Maudsley's* experience leads him to lay stress on sexual excess, and chiefly that carried on systematically by faithful married persons, as a very fertile cause of general paralysis. In other quarters this view has been carried to even a greater extreme.

Having for years sought, and usually in vain, for a history of sexual excess in my own cases, I do not hold with the view that excessive frequency of sexual intercourse, and especially in married life, is by far the most fertile cause of general paralysis. No doubt in some cases, and particularly among the newly-married, this is the cause, in others one of the several causes, of the disease. But I venture to submit that it is erroneous to pay an almost exclusive attention to this cause, as has been done by some authorities on the subject. Certain of whom have gone so far as to assert their belief that when not due to excessive sexual intercourse, general paralysis owns another form of sexual evil, namely masturbation, as its exciting cause. As if, forsooth, the life, both marital and non-marital, of men was but as an orgy of satyrs, either consumed with secret lust, or fitly partnered in salacious revelry by bacchantes lascivious of eye and wanton of limb!

* "Pathology of Mind," London, 1879, p. 433.

When concerned in the causation of general paralysis sexual excess almost invariably, I think, acts in concurrence with other causes,—it then forms but part of that general sensuality and fastness which so oft incur this dread disease. Intense and protracted moral emotion, usually of a painful nature, on the mental side; and alcoholic excess, or exhausting labour sustained by alcoholic stimulation, on the physical side, are apparently more potent and frequent causes of general paralysis than is sexual excess. In the majority of cases where sexual excess has been observed in one who has become a general paralytic, if the research into the antecedents of the patient can happily be carried far enough something else unusual in the demeanour, action, or mental state will be traced to the same period as the sexual excess, and will assist to establish that excess as a prodromic symptom, and not as the original cause of general paralysis, although it then comes to react unfavourably upon the disease, and hasten its progress.

Of the utmost importance in the determination of this matter is the *age* at which the disease is most prevalent. Nowhere else, save partially in the classical work of Bayle, have I met with anything similar to this view—a view previously and independently formed by myself. Most of the cases of general paralysis occur between the ages of thirty and sixty years; it is particularly rife in the decade between forty and fifty; and also very rife between fifty and sixty, relatively to the smaller number of persons in the general population alive at the latter age. Even allowing an appropriate interval between the incidence and action of the cause and the commencement of the disease, it would seem that the age-period of greater frequency of general paralysis is much later than it would be if sexual excess was indeed so potent a cause as is alleged by some.

For at what period of life is it that sexual excess is most frequent? Although many exceptions present themselves, this period may for the most part be assigned to the interval between the age of twenty and that of thirty years. The male sex only is referred to, and, indeed, it comprises the great majority of general paralytics. As a rule after the latter age, or thereabouts, the sexual instinct is less urgent and less sportive, the tempests of desire no longer perturb the being to the same degree as formerly they did,—the blood, which danced so wildly in the veins of passion, now moves in a quiet and easy-flowing, if not sluggish, stream. The whole domain of feeling and hence that of action, too, is sobered by the now more distinct recognition of the realities, and by the burden of the cares, anxieties, and responsi-

bilities of life. Thus the period of life at which sexual excess, and that at which general paralysis, is most frequent are far from corresponding the one with the other.

(γ.) *Moral Causes.*—But if the age at which general paralysis is most rife is not that at which sexual excess is most common; to the incidence of what possible causes of general paralysis is the age in question most exposed? And we may specify the period from the age of thirty or thirty-five to that of fifty-five as the one more particularly referred to. This is especially the age of ambition, pride, selfishness, of speculation, of daring attempts on the heights of fame, of wealth, of power, prestige, and social position; the age of excessive and protracted intellectual labour done under emotional strain, of anxious and sustained strenuous efforts to provide for, and ensure future success to, a growing and exigent family; the age in which excessive physical labour is often undertaken, which, like the intellectual labour, may be sustained by too liberal potations,—and which, like it also, is no longer counteracted by the elastic power of accommodation of youth, or by its restfulness after fatigue. Then, as a necessary correlative, it is the age most liable to chagrins and mortifications of spirit, to losses, to disappointments; the age, occasionally, of sudden beggary after a life of toil and hard-earned success; the age at which, so often, the mirage of hope vanishes and its aerial castles dissolve; at which the projects of life fail and crumble away, and its fire dies out. Then, also, is it, too often, that the spirit of the man wanders forlorn amidst the desolated groves of his affections, the desecrated temples of his noblest aspirations.

No wonder that causes such as these, acting upon those whose nervous systems have lost the elasticity of youth, whose blood and blood-vessels, perhaps, are further impaired by the effects of alcohol, whose naturally hyperæsthetic brains have been exhausted by irritable reaction to every strong impression, no wonder, I say, that in them, causes such as these should bring about a sudden or protracted hyperæmia, a slow irritative form of degeneration, or even of inflammation, in the supreme centres of the organ of mind.

Now superadd to these causes the frequently associated alcoholic excess, and you double their efficiency.

Already noticed as a predisposing cause, mental overstrain acts perniciously as an exciting cause also. Severe intellectual work carried on too protractedly and monotonously, and especially when carried on in an atmosphere of worry, vexation, annoyance, anxiety, may alone produce general paralysis. Emotional overstrain, whether the feelings which become passions and take

possession of the life are of an expansive or depressive sort, may arrive at the same effect. Choleric, "passionate," outbursts often repeated; protracted strainings of ambition in all its phases; the lifelong struggles, chagrins, and heart-burnings accompanying the modern conflict for existence, for place, power and prestige; domestic unhappiness, or bereavements; business anxieties or losses; all conduce to general paralysis. In all cases the original and acquired cerebral power of resistance must be kept in view: of two persons one passes unscathed through mental trials that leave the second in a state of moral and physical wreckage. Hence the immense importance of the predisposing causes.

Moral agony, therefore, to use the phrase of Austin, is a very potent cause of general paralysis. But I would stop far short of his sweeping and exclusive statement that "the complexion, the age, the social condition, the habitat, and the sex of the majority of general paralytics all favour the idea that moral agony is the cause of the disease" (*op. cit.*, p. 88). Surely, the penultimate *the* should be *a*.

Conolly found moral causes in sixty out of ninety-six cases. (*Lancet*, 1849.)

Not seldom, however, does it happen that acts springing out of the intellectual enfeeblement and moral and affective perversion of the incipient disease—together with the turmoil, trouble, and distress they entail—are mistaken for the causes, instead of what they are, the consequences of the disease. See also F. Darde, "Du Délire des Actes dans la Paralyse Générale" (1874, p. 9).

Finally, among other exciting causes, special mention may be made of—

Cranial injuries.

Insolation: not uncommon among British soldiers in India.

Pellagra.

Exposure to great furnace heat. (Calmeil, *Op. cit.*, 1826, p. 374, and *Op. cit.*, 1859, T. i. p. 270.)

Suppression of certain physiological excretions, or pathological discharges.

That general paralysis may be a sequel of various acute pyrexia and phlogoses is suggested by Voisin on very slender grounds. His observations on general paralysis consecutive to protracted generalized neuralgia are better founded. (*Traité*, &c., pp. 333 & 342.)

CHAPTER VII.

MORBID ANATOMY.

A. MACROSCOPICAL—B. MICROSCOPICAL.

A. THE NAKED-EYE APPEARANCES.

These will be so amply illustrated by the cases detailed in the concluding part of this work that a brief synopsis will suffice here.

In some cases where death occurs during the early stages, the brain may even appear to be increased in volume as well as in vascularity, thus narrowing the sulci and distending the dura-mater.

But almost invariably the brain comes under examination at a much later period. The following are then the most marked and frequent naked-eye appearances, they are not all present in every case, and are variously grouped and associated.

Removed from the calvarium, the encephalic mass is usually more or less flaccid, and sinks under its own weight. Then, examining the parts from above downwards, the internal surface of the dura-mater is sometimes found to be partly lined by irregular false membranes, or by russet-hued relics of slight hæmorrhage, to which, in some cases, single or double arachnoid cysts may be added. The dura-mater itself may be thickened and hypervascular, but its alterations are much less frequent and marked in my own experience than they are stated to be by Griesinger and others.

The cerebral layer of the arachnoid is generally thickened and more opaque than usual, and oftentimes changed in consistence—particularly over the fronto-parietal convexity and internal surface of the hemispheres: the opacity often being patchy, and generally more obvious where it covers the anfractuosities and streams along the borders of the meningeal veins. Above, on its outer surface, as first described by Bayle, it is often beset with minute pearly granulations: beneath it, at non-adherent parts, lies serum in the meshes of the pia-mater and in the unduly-rounded anfractuosities of the surface.

The pia-mater itself thickened, coarse, usually hyperæmic

either universally or in irregularly distributed patches, and more or less bathed in serosity especially over the fronto-parietal regions, is also, with rare exceptions, adherent to the summits of some of the gyri. The areas of adhesion are irregular, scattered, and of most variable extent. The adhesions occasionally, though rarely, invade the declivities of the anfractuosities also.

The grey cortical substance of the cerebrum is usually diminished in consistence; much more rarely is it indurated locally, or even rather diffusely:—it is discoloured, is generally the site of hyperæmia of more or less irregular distribution, yet is sometimes wan, faded, and anæmic; is often wasted in certain regions, and perhaps more friable and opaque than normal, and its stratification is oftentimes indistinct. Where adherent to the membranes its superficial layers strip off from the summits of the gyri along with the meninges when the latter are removed, leaving an irregularly eroded, and usually more or less reddish, appearance of the convolitional surface, locally. This is the most characteristic of the gross lesions of general paralysis. The earlier observers, as Bayle, and, seemingly Delaye, attributed these adhesions to arachnitis, the inflammation extending here and there to the brain, though Foville in 1829 denied that they were the simple effect of an inflammation of the arachnoid, and believed that their occurrence at the *summits of the gyri, only*, was owing to the cranial compression to which the summits are exposed during inflammatory turgescence of the brain, aided by the influence of the arachnoid in increasing the thickness of the meninges at those points. Wedl attributed these adhesions to penetration of the cortex, to some depth, by the increased, grouped, nuclei of the adventitious membrane of the vessels of the pia-mater (arteries and veins).^{*} The special adhesive change and its effect I† have termed “adhesion and decortication.” This adhesion of the membranes to the cerebral cortex was found by Parchappe in thirty-nine out of forty-four cases of general paralysis; by Calmeil in seventy-four out of eighty-two cases; by Sankey in eight-fifteenths of his cases; and by myself in forty-five of my last fifty necropsies. Much smaller proportions than these are probably unreliable, as that given by Justin, namely thirteen out of thirty-three. In general paralysis the cerebral grey cortex in my experience is most diseased, as a rule, in the superior and external surfaces of the frontal lobe; next in

* See also L. Besser, of Siegburg, “Allg. Zeitschrift für Psychiatrie,” 1866.

† *Journal of Mental Science*, April, 1878, p. 29.—*Ibid.* Jan. 1876, pp. 571-6.

degree in those of the parietal, and next in the temporo-sphenoidal; while the occipital lobes, comparatively, escape.

The medullary substance is discoloured, sometimes diminished, at others increased, in consistence,—induration being far more common in the white than in the grey substance. Generally hyperæmic, yet when increased in consistence it is often of an unnatural whiteness, and then also displays a *gruyère* cheese, or much-perforated, appearance.

In the more advanced cases there is a decided atrophy of the brain, with increase of fluid in the ventricular, subarachnoid, and arachnoid spaces; for, as the brain shrinks, the various cerebral ventricles become larger both relatively and absolutely, their ependyma, beset with granulations, assuming a pearly, sanded, jewelled appearance, which often is more particularly manifest in the fourth ventricle.

Nor does the grey matter of the ganglia at the base of the brain escape. Changes may be mentioned in the opto-striate bodies which are often withered and pale on the surface, softened or hyperæmic internally: and also in the commissural textures, as the fornix. Austin (*op. cit.*) drew special attention to the alterations occurring here within a circumscribed limit, and principally affecting the optic thalami, soft commissure, fornix, septum lucidum, corpora albicantia, floor of third ventricle, tuber cinereum, and crura cerebri; these alterations often being accompanied by increase of the ventricular serum. In the optic thalami, he asserted that, one or other of the following changes were always to be found, (1) disorganization or softening,—(2) degeneration, with or without induration,—(3) atrophy (simple),—and (4) mere alteration in vascularity without structural lesion.

The pons Varolii and medulla oblongata often participate in several of the general encephalic changes. The granulations of the floor of the fourth ventricle have already been described.

The cerebellum is generally diminished in consistence, somewhat hyperæmic, its tunics, often moderately thickened or opaque, are sometimes adherent to parts of its surface. It has been said by Dr. W. H. O. Sankey (Lectures on Mental Diseases, 1866), that in general paralysis the cerebellum is often increased in weight, that of the cerebrum being diminished. But although its ratio, *relatively* to that of the wasted cerebrum, is generally increased, I have never found a marked *absolute* increase of cerebellar weight in general paralysis, and this experience coincides with recent statistics on the subject, by Crochley Clapham in the 6th volume of the West Riding Asylum Reports.

The meninges of the spinal cord are often thickened, opaque,

hyperæmic, especially posteriorly; the cord itself softened; or, less frequently, indurated, or softened in parts and indurated in others; while it may be surrounded by an undue amount of serum, and it, or its meninges, may bear traces of old capillary hæmorrhages.

There is not one of the above morbid alterations that may not be absent in a given case, but I have never made the necropsy of a general paralytic without finding very obvious naked-eye changes in the cerebro-spinal nervous system and its protecting tunics. The view of Lélut, Aubanel and Thore, that general paralysis might occur and run its course without any alteration of the brain and meninges, was no doubt the result of faulty, imperfect, and non-microscopical observation (L. V. Marcé, *op. cit.*), or of diagnostic error. Indeed, Bayle asserted that the following lesions were always found in general paralysis:—the arachnoid more or less opaque, thickened, increased in consistence and density, and containing serosity; the pia-mater more or less injected with blood and œdematous. These lesions occupied the convexity and internal face of the hemispheres, and to them he added those of the ventricular arachnoid, which was thickened, unduly tough, and beset with granulations. Other, but varying, lesions were also usually present. By Salomon the increased opacity and condensation of pia-mater and arachnoid, and the soft, pappy, and unduly loose state of the cerebral cortex, were declared to be constant in general paralysis.

With reference to the amount of atrophy of brain in general paralysis we may turn to the brain-weights.

The following were the *average* brain-weights of 197 *male* general paralytics of all ages and of 46 *female* general paralytics of all ages, reported by Crochley Clapham. (West Riding Asyl. Rep. vol. vi. p. 21.)

| | Ozs. | Grammes. |
|---|----------|----------|
| Average weight of entire encephalon of <i>male</i> general paralytics | 45·92 or | 1302·015 |
| Average weight of cerebrum of <i>male</i> general paralytics | 39·66 „ | 1124·5 |
| Average weight of cerebellum, pons and med. obl. of <i>male</i> general paralytics | 6·26 „ | 177·5 |
| Average weight of entire encephalon of <i>female</i> general paralytics | 40·01 „ | 1134·304 |
| Average weight of cerebrum of <i>female</i> general paralytics | 34·39 „ | 975 |
| Average weight of cerebellum, pons, and med. obl. of <i>female</i> general paralytics | 5·62 „ | 159·3 |

In these cases the meninges, or portions of them, and a certain amount of serum were, probably, weighed with the nervous

masses. Possibly, the "weight of the entire encephalon" as given in these tables was taken immediately upon its removal from the skull, in which case the pia-mater, visceral arachnoid, subarachnoid and ventricular serum, would all contribute to swell the total weight.

But it is the weight of nervous tissue that is required, and therefore I append the average weights in forty of my cases, in which the brains were stripped before being weighed, a few shreds of meninges only remaining, and that only sometimes, and especially on the cerebellum, pons, and medulla oblongata. These general paralytics were males (soldiers), and most of them aged thirty to forty

| | Ozs. | |
|---|-------|--|
| Average weight of entire encephalon . . . | 44.15 | |
| " " cerebrum . . . | 37.79 | |
| " " right cerebral hemisphere . . . | 19.04 | } not ascertained separately in all |
| " " left " " . . . | 19.17 | |
| " " cerebellum . . . | 5.42 | |
| " " pons Varolii and medulla obl. . . | 9.45 | |
| Maximum weight of cerebrum . . . | 48 | |
| Minimum " " . . . | 25½ | |
| Maximum " " (right hemisphere) . . . | 24½ | |
| Minimum " " " " . . . | 12½ | |
| Maximum " " (left hemisphere) . . . | 23½ | |
| Minimum " " " " . . . | 13 | |
| Maximum " cerebellum . . . | 7 | |
| Minimum " " . . . | 4½ | |
| Maximum " pons Var. and med. obl. . . | 1½ | |
| Minimum " " " " . . . | ¾ | |

We may compare the above average weights in my own cases, all being males, with those given by Dr. R. Boyd (Philosophical Transactions, 1861, p. 241), for the male insane of all kinds dying between the ages of thirty and forty at the Somerset Asylum. Unfortunately the effect of general paralysis can only partially be traced in this way, inasmuch as some of these cases, also, were general paralytics. In males aged from thirty to forty years and subjects of the various kinds of insanity he found the average weight of the right cerebral hemisphere to be 19.82, of the left 19.94, of the cerebellum 5.33, and of the pons Varolii and medulla oblongata 1.05 ozs.

Or they may be compared with Dr. John Thurnam's return of the *average* weights of insane males between thirty and forty years of age at the Wilts County Asylum; namely, cerebrum 39 ozs. (1,105 grammes): cerebellum, pons Var., and med. obl., 6 ozs. (170 grammes). (Journ. Mental Sci., Apr. 1866, p. 34.)

Or with Crochley Clapham's (*loc. cit.*, p. 26) figures for in-

sane males of all kinds æt. thirty to forty; viz.: cerebrum 40·9 ozs. (1,161 grammes): cerebellum, pons, and med. obl., 6·8 ozs. (180 grammes).

Or we might compare them with the average weight of the *entire encephalon* of sane males of the same decade;—namely, 49 ozs. (Thurnam, *op. cit.*, p. 40.)

The wasting would thus appear to be from one-and-a-half to three and five ozs., but the reckoning is somewhat vitiated by the fallacies above referred to; namely, the imperfect separation of meninges, and the indiscriminate character of the statistics quoted.

On the other hand, the average brain-weights of healthy soldiers would probably exceed the average brain-weights of healthy males of the lower class and of the districts from which the above instances, compared with the soldiers, were drawn, and if so the brain-atrophy in the military general paralytics would *pro tanto* be really greater than appears.

Lunier held that of the three structures,—meninges—grey substance—white substance—all, or any one alone, may be affected in general paralysis, and that no one lesion is constant.

Of late years much attention has been devoted, and much importance assigned, to the spinal lesions in general paralysis;—unduly so in my opinion. Yet, especially when there have been symptoms of “ataxia” or paraplegia, has Tamburini often found osteomata on the spinal arachnoid.

It would be tedious to describe at length a number of morbid changes to which prominence has been given by various writers on general paralysis. We may briefly glance at a few.

Delaye described as the principal changes, (1) induration of the white cerebral substance: (2) infiltration and adhesion of the meninges to the brain, which then is softened: (3) shrinking and atrophy of the brain, and increase of intra-cranial serosity.

The elder Foville, followed by Legalle-Lassalle, spoke of alterations of the white substance, constantly present but varying in kind, and undue adhesion of its planes: induration and pallor of the superficial layer of the grey cortex, and softening of the sub-jacent layers.

Softening of the middle layer of the cerebral grey cortex, and cerebro-meningeal adhesions, were described as the most characteristic changes by Parchappe; and indurated, irradiating, tuft-like expansions in the white substance, just beneath the grey, of the cerebral convolutions, by Baillarger and Regnard;—inflammation, usually causing softening, and invading the cerebral hemispheres layer by layer from the periphery to the central parts, by

Belhomme;—cortical softening and cerebro-meningeal adhesion by Bottex;—induration of the septum lucidum by Conolly;—cortical softening of the anterior cerebral lobes by Falret;—greyish-red softening, or colouration, and partial superficial induration of the brain cortex by Griesinger;—extreme atrophic attenuation of the anterior part of the cerebral grey cortex by Erlenmeyer, Bonnet, Poincaré, and Hitzig;—and induration of the grey substance, of the nature of sclerosis, by Frerichs;—softening, hyperæmia, serous infiltration, vascular changes, and yellow zones, all of the *cerebellar* cortex, by J. Luys;—hard and cicatricial state of *lamina nervea* of *cerebral* cortex in final stage by Salomon;—morbid change of *outer* layers of cortex by Clouston, also. Let us add that Baillarger and Baume speak of considerable inequality in the weights of the two cerebral hemispheres in some cases of general paralysis; inequality of which kind was connected by Follet in an especial manner with epilepsy. Discovered by Brunner in 1694, the granulations seen on the ventricular walls of the brain, especially in the fourth ventricle, and first and very fully described in general paralysis by Bayle (1826), and mentioned by Calmeil (1826), and Daveau (1830), occupied the attention of Rokitansky and Virchow in 1862, and in 1861 were the objects of an imagined discovery by M. Joire, who erroneously viewed them as invariably found in, and special to, general paralysis.—(Rep. Acad. Med., 1861 :—and Gaz. Médicale de Paris, 1864, p. 528.)

Passing to other parts and tissues of the body:—

Ears.—One or both ears may be found atrophied and distorted as a sequel of othæmatoma.

Bones.—Dr. E. L. Ormerod (Jour. Mental Sci., Jan. 1871, p. 571), speaks of the bones in one case of general paralysis as being “dark, singularly wet, and greasy.” The internal osseous tissue was absorbed, was replaced by an excessive deposit of the fatty matter normally existing in its interior,—the whole bone was affected, also, by obscure disintegration of the osseous structure itself, and by “infiltration of oily matter into the substance, which had intruded itself within the Haversian canals.” Another case is mentioned by Dr. I. Ashe (Jour. Mental Sci., April, 1876, p. 82), in which “in the bony structure of the ribs the Haversian canals seemed almost obliterated by a mass of degenerate deposit containing oil.” Bones like these would break with fatal facility. In this case, also, the muscular structure of the heart and of the gastrocnemius was reported as being pale and fatty.

A case in point is mentioned by Dr. S. W. D. Williams. (Jour. Mental Sci., April, 1878, p. 161.)

In another general paralytic, one under Dr. H. Bonnet, the humerus was fractured and fissured by a fall. Its medullary substance was a mere "bouillie sanguinolente." (Biante, Ann. Méd. Psych., Nov 1876, p. 350.)—Thinning of the compact tissue of the bones and proliferation of fatty elements in both the medullary, spongy, and hard portions were found in several cases of this kind.

One general paralytic under my own care fractured a metacarpal bone by a blow of his fist against the side of a padded room; and afterwards, when walking by himself, twisted his leg in turning, and fractured a fibula. Others have fractured their own bones in attempting to escape; and probably the bones of many general paralytics are unduly brittle.

Cases such as those just mentioned are of special interest with reference to the bone-fractures incurred by general paralytics when violence on the part of attendants is alleged or suspected.

General Frame; Viscera, etc.—The following particulars are entirely derived from records of necropsies made by myself on general paralytics.

It is unnecessary to reproduce the figures relating to several conditions; such as the *emaciation* which, usually present in some measure, is often extreme; the *cadaveric lividity*, and the abdominal discoloration, as to which there is nothing very special: while *bedsores*, *blebs*, and *boils*, are observed with conspicuous relative frequency.

Rigor Mortis (all seasons, twenty-four to forty-eight hours after death).—In about one-third of the cases my notes describe rigor mortis as being "marked" or "considerable," in another third as being "moderate," in about one-quarter as being "slight," and in about one-ninth as being "absent."

Contraction of the Limbs, especially of the lower ones, is occasionally observed.

Heart.—Pericardial fluid somewhat increased in one-third of the cases. Patchy fibroid thickening of the visceral pericardium is occasionally seen. Blood: usually the right chambers of the heart are full, the left auricle moderately full, the left ventricle nearly empty and more or less contracted. The cardiac clots are softish, occasionally firm, rarely is the blood entirely fluid. The heart-muscle is more or less softened and unduly friable in about two-thirds of the cases. Usually of dull aspect, its hue is sometimes pale or slightly yellowish, occasionally darker. One or both of the *valves* of the left side of the heart are altered in about one-third; increased thickness, opacity, atheromatous and calcareous changes, are by far the most frequent.

In more than half there is slight "atheroma" of the *aortic arch*;—in a few others there is marked "atheroma," especially of the form "endoarteritis deformans." In more than half of the cases one or both of the *coronary arteries*, especially the left, are more or less atheromatous.

The *average* weight of the heart in my experience was 10½ ozs. This is below the average weight stated by Burman for all male general paralytics, but approximates that given by him (10·95 ozs.) for male general paralytics dying in the same decade—thirty to forty—as did most of my cases.

Lungs.—Old pleuritic adhesions or pleuritic thickenings in two-thirds. Hypostatic congestion, or marked congestion and œdema of bases, in more than two-thirds. Anterior emphysema in half. Some serous fluid in pleura in nearly half: much watery or frothy secretion in bronchi in one-third: occasionally, enlarged bronchial glands: calcareous nodules from former obsolescent caseation in one-sixth: fibroid change and cirrhosis, limited to one apex, or to both apices, in one-sixth. The foregoing changes are either chronic, quiescent, and comparatively innocuous, or connected with the mode of dying.

But active, and even grave, disease of the lungs is not uncommon in general paralysis. In one-third of the cases there was more or less pulmonary tuberculosis, occasionally there was ordinary caseous (catarrhal) phthisis; in one-third, marked hypostatic pneumonia; and, in another one-fourth, a form of lobular pneumonia; both, occasionally, found with or passing into slight local gangrene: in one-sixth, recent pleurisy. Average weight of right lung 31·78 ozs., of left, 28·61 ozs. The right lung was the heavier in 78 per cent., the left the heavier in 22 per cent.

Stomach and Intestines.—The mucous membrane of the stomach, occasionally pale, was often the seat of patchy hyperæmia, with passively distended veins. The same was seen at times in the small intestines, and the mucous membrane of the colon was sometimes thickened or ulcerated, and in some cases the colon was somewhat loaded with feces.

Liver.—In two-thirds of the cases there was marked passive congestion of the hepatic veins and sublobular radicles and centres of acini. The hepatic substance, unduly friable in some cases, was less often firm and even slightly cirrhotic. Average weight 57 ozs.; maximum, 84½ ozs.; minimum, 44 ozs.

Spleen.—In one-third, the spleen was decidedly too firm: in a few, unduly soft. Old perisplenitic adhesions, old cicatrices, old general capsular thickening, or a local thick hard cartilaginoid

patch in the convexity of the capsule, were each observed in a few cases; and now and then the spleen was unusually notched or fissured. Average weight, $6\frac{1}{2}$ ozs.; maximum, 12 ozs.; minimum, $2\frac{3}{4}$ ozs.

Kidneys.—In one-third, undue adhesion of the capsules, the adhesion usually being slight; occasionally granular kidney; in one-fifth some cystic change of the ordinary kind.

Surgical nephritis occurred in a considerable number towards the close of life, and usually was accompanied by pyelitis. Now and then renal embolism was observed. Congestion of the kidneys was not infrequent. Average weight of right kidney, $5\frac{1}{2}$ ozs.; of left, 6 ozs. In one-fourth there was some abnormality of the renal arteries, usually consisting of an unusual point of entry or a double renal artery: once I found renal calculus, the affected kidney being also atrophied and cirrhotic.

The *Bladder* sometimes showed traces of chronic or sub-acute cystitis, or of slight mucous or submucous ecchymosis.

B. THE MICROSCOPICAL APPEARANCES IN GENERAL PARALYSIS.

If practicable, it would be highly desirable to trace the entire series of changes undergone by the several constituents of the nervous organs in various cases of general paralysis. But such a history would be more or less conjectural in direct proportion to its exactitude. Observers, varying in degrees of skill and judgment, have caught glimpses of the morbid processes at points of their course, and after the recital of some of these observations I will briefly refer to my own. So great is the variety of morbid appearances described, and of localities said to be principally affected, that I propose to trace the changes throughout the whole central nervous system; beginning at the brain, as a matter of convenience, and without prejudice to the question as to what part of the nervous system is the primary or principal seat of the disease,—a question as to which the most conflicting replies have been returned by workers in nervous pathology. With facility one might dogmatize; but, in the present imperfect and shifting state of our knowledge, I think it better to quote several observations, even at the risk of being thought tedious.

The Cerebrum.—The microscopical appearances in the cerebral convolutions, especially in their cortical grey matter, will be described under three heads as they affect, (1) the blood-vessels of the cortex, (2) the neuroglia, and (3) the nerve-cells and fibres.

1. THE BLOOD-VESSELS OF THE GREY CEREBRAL CORTEX.

The microscopical condition of the minute blood-vessels of the cerebral convolutions varies very much in general paralysis. It is influenced by many conditions which differ in each case. Among these modifying conditions may be enumerated:—(a) The age of the patients. (β) The presence or absence of degenerative vascular changes independent of any connected with the morbid process causing mental disease, and due to causes such as alcoholism or renal disease.—(γ) The duration of the general paralysis.—(δ) The mode of preparing the sections.—(ε) The sites from which the specimens are taken; the vessels often being much more diseased in some cortical regions than in others, or their morbid changes, occasionally, differing in kind, also, in various regions. Hence it is that wide divergencies are found in the descriptions given by several observers of the morbid vascular appearances in the brain in general paralysis. And what is true of the vessels in this relation applies, in part, also to the nerve-cells and to the neuroglia. I will here briefly summarize a few of these observations.

The researches of Wedl did not refer solely to general paralysis, and are of but limited value for our purpose. In the vessels he described irregular hypertrophy of their walls, wavy longitudinal markings, transverse ridges becoming brownish and more wrinkled; round the small arteries, capillaries, and veins a growth of hyaline embryonic connective tissue beset with oblong and grouped nuclei; which becomes more fibrous, while about it fatty or calcareous and pigmented granules may appear. The growth of connective tissue in the vascular walls may also lead to compression and obliteration of the capillaries.

Mention will be made of Rokitansky's view that the primary and essential change in general paralysis affects the cerebral connective tissue. He described this increased growth of connective tissue as beginning, partly at least, upon the walls of the capillaries, which latter were also enlarged, twisted and doubled, or presented aneurismal bulgings.

That in general paralysis the contractility of the arterioles was sometimes destroyed by amyloid degeneration, was the view of Tigges.

W. H. O. Sankey found the capillaries of the brain-cortex tortuous, serpentine, or even sharply curved or kinked, or forming as it were varicose knots or vessels. The varicosity of the capillaries of the cortex he found to be extremely common, if not constant. He also depicted a hyaline substance around the vessels, to the contraction, and perhaps fibrillation, of which he

supposed the vascular tortuosity to be due. But this in truth was the perivascular lymph-sheath of Robin and His, and therefore a physiological condition.

The highly developed capillary network of the cortex, especially of its inner layers, was described by Franz Meschede as being crammed with blood-corpuscles, elongated, and presenting points of extravasation, and the minute vessels as being sometimes dilated, atheromatous, or fatty.

Lockhart Clarke found the investing sheaths of the vessels, especially in the white cerebral substance, to be thicker and darker than normal in general paralysis, and often to appear like fusiform dilatations along the course of the vessels. Hæmatoidin granules were also abnormally abundant. To which we may add the radiating series of streaks and lines in the grey and white substance; and the vertical fissures and oval slits, containing blood-vessels and more manifest in the white substance; as well as the vacuoles described, elsewhere, by the same observer in the white substance of the cerebral convolutions, but occurring also in the optic thalami, pons Varolii, and anterior pyramids of the medulla oblongata—vacuoles of round, oval, crescentic or somewhat cylindrical shape; in size from that of a grain of sand to that of a pea; having no lining membrane, and being sharply cut out of the tissue—vacuoles which are empty, or contain blood-vessels, or *débris* of blood-vessels, or hæmatoidin, and which probably represent perivascular spaces which originally contained blood-vessels surrounded by their peculiar sheaths, and which subsequently became empty by the destruction and absorption of those vessels.

The minute cortical blood-vessels were rigid and sticking out like bristles in a young general paralytic examined by S. Wilks.

Some years ago H. Schüle described, both in the brain and cord, abundant proliferation of nuclei in vessels both internally and externally, aneurysmal degeneration, amyloid degeneration, obliteration and new formation of vessels. Very recently he states that in the earlier stages of the disease he found hyperæmia, especially in the internal layer of the cortex; dilatation of the vessels; thickening of their walls and increased number of nuclei, also an increased number of Boll's pencil-cells. In the terminal stage he found the vessels blocked by sclerosis and proliferation external to them, and the lymph channels dilated.

Of four cases described by Major, in the first and third the cortical vessels were dilated but not thickened, and were the seat of abundant hæmatoidin deposits; some were very tortuous; in the second were hæmatoidin deposits, and the vessels were frequently

looped, varicose, and tortuous; and in the fourth the vessels were dilated, and had copious deposits of nuclei and pigment.

Previously described by L. Meyer and by H. Schüle, the development of new capillary vessels in the brain in general paralysis was also asserted by A. Lubimoff. Immediately about minute spots of softening in the frontal lobes of a general paralytic, and as the investigation approached the softened centres, he observed the stellate cellules of the interstitial tissue become more and more numerous, those of their branches attached to the vessels becoming larger and larger, and either taking the direction of the capillary and enlarging progressively so as to be confounded with it, or becoming radii as if from a centre of capillary ramification,—or, again, anastomosing with branches of other cellules to form a capillary vessel.

J. P. Gray appears to view the primary changes as occurring in and about the adventitious sheath of the arterioles and large capillaries. He speaks of thickening and hyaline infiltration of the walls of small vessels; accumulations of lymphatic, fatty, and pigmentary elements from irregular distension of the sheaths by capillaries; or these last as being surrounded by a dense layer of nuclei or cell-bodies of spongy-like structure.

On the other hand, and in opposition with several of the observers just mentioned, Bonnet and Poincaré deny that proliferation of cellular tissue around the blood-vessels is ever so marked as to reduce or obliterate their lumen. In the arteriolar parietes they found brown pigment, yellow crystals, and fatty granulations: and the same pigment and crystals, within the (Robin's) sheath of the capillaries; while vast collections of fatty granulations sometimes surround portions of the vascular walls; and within the vessels themselves free fat-globules are found.

Ludwig Meyer held that the first changes in general paralysis occur in the capillary vessels of the grey cortex—that a chronic inflammation begins there. A proliferation of cells occurs around the walls of the vessels. These are full of formed cells, and not of degenerated nuclei as he once believed; occurring in clustering patches, the latter may pierce the internal wall of the vessel and obliterate it; or may bulge into the vessels or make their lumen disappear, by pressure from without. These changes he traced down to the medulla oblongata.

Thus is the circulation interrupted, and under the effect of the sanguineous pressure in neighbouring permeable vessels ectasies and varices—true microscopical aneurisms—form. These explain the hæmorrhagic-like foci seen in the brain-sections in general paralysis. The alteration first of all attacks the grey substance,

later on, invades the white, and in the final periods does not spare the parts at the base, as the opto-striate bodies and pons. At last, portions of the cerebral substance become atrophied, either by pressure, or by an ever-increasing failure in their circulation, and the organ enters into the phase of fatty degeneration with production of nucleated cells.

There also occur fatty and calcareous degeneration of the vessels, widening of their calibre, and the formation of minute aneurisms, often of the dissecting variety.

The observations of Mierzejewski include a great variety of changes in the vessels. At first there is nuclear hyperplasia in the capillary walls, and sub-adventitial assemblage of nuclei (cells according to L. Meyer) in the smaller vessels. Later on, are miliary aneurisms, sanguineous extravasations with vascular rupture, the capillaries and smaller vessels being thickened and homogeneous, and their walls of glass-like aspect. At last comes general fatty degeneration of the vessels.

The formation of new capillary vessels beginning as buds or appendices on the old capillaries is described: the increase of nuclei takes place in all the coats of the vessels, but particularly affects the round nuclei of the adventitia: there is wandering of leucocytes and red blood-globules into the sub-adventitial space of the vessels: the retrograde changes in these may excite proliferation of the nuclei of the vascular walls, impair their elasticity, and lead to miliary aneurism.

To continue his description. In the adventitia chronic inflammatory processes and pigment deposits are always present. The vessels may become twisted and serpentine as they lie within their adventitial sheaths; their fatty degeneration may be primary or secondary, and the capillaries may put on a spiny or villous appearance, either owing to fine appendices, or to attached and ruptured bundles of fibres, or to the adhesion of cells of interstitial tissue to the vascular walls. Slight blood-extravasation may occur; rarely, rupture from fatty change.

According to Hitzig the nuclei of the vascular walls in the brain are increased in number, masses of cells adhere to them filling the perivascular space or dilating it, or wandering into the parenchyma. These, probably, are white blood-corpuscles. Here and there, also, red blood-corpuscles undergoing pigmentary changes fill the lymph-spaces for a distance. The vessels are crowded with blood-corpuscles, and their fatty or amyloid or colloid degeneration is stated to exist, the nerve-cells participating in the same changes.

In a recent description by J. Luys the outer coat of the minute

vessels is said often to be in a state of connective-tissue proliferation, whereby they are frequently constricted at points, or dilated, or made moniliform at others, in consequence of the unequal action of the new growth on their walls—a reversion, it will be noticed, to the views held by several of the earlier observers.

Writing in 1875 with reference to dysphasia in general paralysis, Aug. Voisin mentioned in certain cases “hyperæmia, infiltration with blastema, with exudation and recent embryoplastic production, and softening, of the cortical layer of the anterior lobes of the brain.” The vessels present in their walls, and in the spaces by which they are surrounded, an abundance of embryoplastic nuclei in very close rows: the nervous substance itself contains a numberless amount of these nuclei. They may also be seen “in the course of the nervous fibres which pass from the cortical substance of the anterior convolutions to the medulla oblongata through the white cerebral substance, the corpora striata, and the pons Varolii. In the course of these fibres and in their interior you meet with an enormous quantity of embryoplastic nuclei which are especially abundant in the vascular sheaths in the perivascular spaces, and which thence certainly have invaded the nervous substance; and you meet, also, with masses of hæmotosine, of hæmatine in yellow or colourless crystals, or effusion of blood corpuscles more or less old.” In his recent work Voisin describes in the brain-cortex, endoarteritis, evidenced by nuclear bodies, rounded or oval, more or less packed, and forming encircling chaplets or muff-like surroundings of the vessels;—perivascular blastemic effusion;—effusion of blood-globules around the vessels, and hæmatine crystals in and about their walls, with hæmotosine granulations in the perivascular lymph-space, which last is often increased in width;—encircling chaplets of fusiform bodies around the vessels, hæmorrhages of the external coat of the vessels and hence compression of the vessel from without;—ampullary dilatations or miliary aneurisms, or compression or obliteration of vessels by the organization of fibrillary tissue from the cellules lining their walls;—and colloid degeneration.

And here it may be added that in the white substance of the cerebrum Voisin found similar changes in the vessels, and a similar production of numerous round or oval embryoplastic bodies in the intermediary substance.

In the cortex of the Insula, the same changes as above described.

In the optic thalami, hyperæmia, clouding of the sections, vessels gorged, but the blastemic infiltration and nuclear overgrowth far less than in the cortex; occasionally local dilatations, or sclerous change of the vessels.

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In the corpora striata, rarely, foci of hæmorrhage or softening.

In the cerebral peduncles, but slight vascular change; at times granulo fatty degeneration of cell-groups. In the geniculate bodies, simple endoarteritis with nuclei and old blood-products in and around the vascular walls. As to the pons; at the nuclei of the third nerves, the nerve-cells fatty, misshapen, notched, and yellowish; the vessels engorged and the source of infiltrating embryoplastic nuclei.

2. NEUROGLIA (microscopical appearances in general paralysis).

Rokitansky described an overgrowth of connective tissue in the grey cerebral cortex; producing a greyish, viscid, tenacious fluid containing nuclei; rendering the cortex soft and succulent, and then becoming stiffer and fibrous, and retracting here and there. To the effects of this growth of connective tissue he traced the changes in the nervous elements.

On the other hand Westphal denied that there was any proof of an increase of the connective tissue of the brain in general paralysis. Yet at the same period we find Magnan describing, as the constant lesion in general paralysis, an abundant nuclear proliferation in the interstitial tissue of the whole brain and on the walls of the minute cerebral vessels;—the morbid change in the interstitial tissue being primary, and that of the parenchymatous elements of the brain being consecutive.—H. Schüle, also, observed abundant nuclear proliferation in the neuroglia, and a dense granular fibrillary condition of its intercellular substance.

In the interstitial substance Meynert and Lubinoff found immense branched (spider) cells; and the latter observer traced them throughout all the interstitial substance of the brain, but especially in the deeper layers of the grey matter of the convolutions.

In general paralysis Mierzejewski divides the neuroglial changes in the *white* substance of the cerebral convolutions into three stadia

In the *first* stadium there is an increase of the nuclei of the interstitial tissue, the nuclei having three principal forms:—(a) Some are round, tinting highly with carmine, having regular outlines, a granular aspect, and sometimes fine spines.—(b) Others of oval shape, colouring feebly with carmine, of single contour, and finely granular aspect.—(c) A third set are oblong in shape, with lateral constrictions or with shining spines on their borders; or of horseshoe shape; these are often seen near blood-vessels.

In the *second* stadium there are, in the white substance, islands of amorphous opaque homogeneous appearance, ill-defined at the margin, of regular form, of unequal rough vacuolated surface;

tinting strongly with carmine, and imprisoning nuclei, or having the latter attached to their borders. Radiating filaments pass from these and form a network in the interstitial tissue; they contract and put on the appearance of stellate cells, but true stellate cells, he says, are rare here. In his belief these islands consist of connective tissue nuclei fused together by coagulated fibrine, and he had seen the same in a hæmorrhagic *foyer*.

In the *third* stadium the nuclei of the interstitial tissue are atrophied, often extremely so, and they become angular.

Hitzig speaks of finding the spider cells, connected with the vessels, in general paralysis.

In some general paralytics J. Luys observed in the white substance of the convolutions, great increase of the fibrillæ of the neuroglia, and swelling of the neuroglial corpuscles with an increased number of their thickened and radiating processes, thus invading and destroying the nerve-fibres, and forming a sclerous closely woven tissue, here and there of areolar appearance.

In the superficial regions of the cortex, also, the neuroglia, cushioned between the meninges and the deeper layers of the cortex, and forming a sort of natural epiderm, undergoes neoplastic proliferation and increase, and its swollen and multiplied corpuscles and their prolongations form trabeculæ, or irradiations, or an intersectional lattice-work; in a word—an invading and sclerose tissue, which, affecting also the nerve-cell-bearing layers of the cortex cerebri, gradually encroaches upon and destroys the nerve-cells themselves. The neuroglial hyperplasia follows the same course here as in the white substance, and the same changes occur in the intervening layers of the grey cortex, these changes, indeed, exhibiting a continuity. The nerve elements, invested, encircled, and compressed, lose their pyramidal outlines and true morphological character, and undergo necrobiosis and progressive resorption. Thus the lesions of general paralysis are distinctly characterized by a generalized hyperplasia of the neuroglial woof. This begins sometimes in the white substance, sometimes in the submeningeal region of the cortex, sometimes in the deeper layers of the same, and sometimes in the spinal cord.

The vessels of the parts, also, are profoundly altered by the new growth. Thus in certain parts the circulation is obstructed and the nerve-cells put out of use, and atrophy of the gyri follows this stifling of the nerve elements.

As to the neuroglia Voisin, following Robin, denies its existence as a connective tissue, and he finds in the intermediary substance of the cortex an infiltration of blastema, by transformation of which a network of nuclei appears along the course of the

vessels, and, therefore, developing from the blastema at the connective woof of the vascular parietes. Of these, the nuclei lying in, and immediately about, the vascular walls may either remain free nuclei, or may become fibro-plastic cellules—cellules of connective tissue—or connective tissue fibrils. Others, developing in the depth of the intermediary substance take on the characters of its normal myelocytes. The myelocytes suffer morbid changes analogous to those of the cellules.

8. THE NERVE-CELLS AND FIBRES OF THE CEREBRAL CORTEX.—Rokitansky believed an overgrowth of the connective tissue of the brain to be of the essence of the affection. He described the nerve-tubules in the grey cortex, pressed upon by this connective tissue growth, as becoming varicose, or broken down into varicose or rounded or club-like or corpuscular elements with fat and granular matter, and their metamorphosed detritus as becoming amyloid or, again, colloid bodies, which, in their turn, finally disappear, nothing but a mere induration remaining. And he described some of the nerve-cells as becoming inflated. Other observers have described colloid and amyloid bodies in the cortex, and even colloid degeneration of the nerve-cells.

Tigges thought he observed an increase of the nuclei in the ganglionic cells of the cerebral cortex in general paralysis.

Parenchymatous inflammation of the cerebral nerve-cells was described by Franz Meschede in general paralysis, and as being the essential lesion. It existed, he said, in every degree, from congestive imbibition and parenchymatous swelling, to fatty and pigmentary degeneration—and reduction to molecular detritus—of the ganglion cells of the inner layer of the grey cortex, and especially in certain regions; these cells in the early stage being softened, voluminous, and isolated.

In one case the nerve-cells were described by S. Wilks as being altered in form and colour, and as apparently containing earthy matter. Amyloid bodies also presented themselves in the superficial grey substance.

Lockhart Clarke found an increase of the pigment granules contained in the nerve-cells, which in some instances may completely fill the latter—or the cell, losing its sharp outline, may look like a heap of particles ready to fall asunder. In other cases he observed some nerve-cells unusually loaded with pigment; some more or less disintegrated; and here and there irregular masses of fatty particles of different sizes scattered over areas of variable extent.

Meynert, in 1868, described a proliferation of the nuclei of the cortical nerve-cells, and a series of changes, viz.—(a) vesicular

transformation of the nuclei:—(b) nuclear division, simple or multiple:—(c) dropsical swelling of the ganglionic nerve-cells, shown by their increased size and hyaline aspect, and by the dark edges of the nuclei:—(d) sclerosis or sclerotic swelling of the cells; their edge being dark; their protoplasm being augmented, homogeneous, strongly refracting;—their contours well defined, sometimes angular or indented;—their prolongations swollen, and appearing more numerous;—and their nuclei invisible, though the nucleoli may perhaps be seen:—(e) molecular destruction of the protoplasm of the cortical nerve-cells, and shrinking of their volume.

Mierzejewski observed various changes in or about the nerve-cells. Thus (if near blood-vessels with sub-adventitial hæmorrhage), there may be increase of the connective nuclei, which then surround the nerve-cells; and sometimes are attached to their surface;—or the cells may be enwreathed by a band of fibrine. The nerve-cells are also encircled by vacuoles, they are increased or diminished in size, their protoplasm is dimmed, and they stain well with carmine. Later, these nerve-cells lose their fibrillar structure, become loaded with brown and yellow molecules, their nuclei lose consistence and disintegrate, beginning at the centre. Filling up with yellow and brown pigment, insoluble in ether, they gradually cease to take the carmine stain. Or, again (if near vessels, or near extravasation external to the vessels), the nerve-cells may be increased in volume, and filled with a pale granular material soluble in ether. Finally, the cells are transformed into bodies replete with granular substance, without a nucleus and failing to take the carmine hue, and their several prolongations undergo granular destruction, that from the axis-cylinder proving the most resistant to change.

In certain parts he also found peculiar bodies—some oval, and some of riband-shape—which he considered to be hypertrophied axis-cylinders, or their remains, but which Hitzig has since asserted to be in reality artificial results of the mode of preparation.

In different cases Major found: I. Some cells loaded with pigment-granules, rounded, of inflated appearance, and their branches and nucleus disappearing; some immense cells in mid-depth of the cortex;—and the nerve-fibres tortuous and irregular in their course.—II. and III. Bodies of the cells shrunken around the large, slightly-staining, nucleus; an irregular inflated appearance of some cells.—IV. Simple atrophy of the nerve-cells.

Granular degeneration, and diminished number of nerve-cells,

fatty and crystalline masses in the grey substance, and "proteinous" bodies in the white substance, of the convolutions, are mentioned by Gray.

The nervous elements of the cortex were described by Voisin as becoming blended with embryoplastic deposits, which ultimately undergo fibrillar change and produce atrophy; the embryoplastic nuclei are also infiltrated among, and in, the fibres radiating from the frontal cortex to the medulla oblongata. Here also may be found deposits of hæmatosine, hæmatine, and old collections of blood-corpuscles. In his recent work he states that the nerve-cells, at first with yellowish protoplasm and scarcely visible nucleus which take the carmine stain but slightly, next become deformed and more fatty, their prolongations suffer injury, and their nuclei cease to stain. Finally, their protoplasm is pigmented, their nuclei are invisible, and their prolongations destroyed. The appearance of the cortex and sections is cloudy.

According to Bonnet and Poincaré the nerve-cells often contain fatty granulations, which form agglomerations about the nuclei or at the commencement of the branches of the cells. These granulations, usually uncoloured, but exceptionally yellow, are void of pigmentary granules. Changes of these kinds occur in all parts, but most abundantly in the frontal lobes. The nerve-cells of the optic thalami and corpora striata are similarly affected; those of the cerebellum and pons are healthy; those of the medulla oblongata may occasionally be fatty, but always have abundant rusty-hued granulations. The nerve-fibres are healthy, they say.

Rutherford and Batty Tuke have observed hypertrophy of the nerve-cells in general paralysis.

Fatty degeneration of the nerve-cells, their pigmentary degeneration, more rarely, their swollen sclerosed state is described by Hitzig. He also treats of appearances supposed due to hypertrophy of axis-cylinders.

The fifth layer of grey matter, according to Spitzka, becomes the seat of sclerosis and its spindle-cells diminish in number: while an absence of any alteration of the cerebral nerve-cells in the early stages of general paralysis is asserted by L. Meyer.

Personal Observations.—My own microscopical examinations in general paralysis have mainly concerned the cerebral cortex, and, concisely stated, the following were the principal changes found.

In the advanced cases fatty particles, free, or in the individual tissue-elements, were sometimes observed on the sections.

The Cortical Nerve-cells.—Sometimes atrophy or shrinking of the large nerve-cells was observed, associated, or not, with the

appearance of vacuoles, surrounding or beside them;—sometimes they were of a dull dimmed appearance, took the carmine stain badly, and their nuclei were obscured;—or, again, granular or fuscous degeneration of the nerve-cells was present in various degrees, occasionally even to disintegration of the cells with destruction of their processes. One or more of these changes, and sometimes others, existed in a given case.

The Neuroglia.—In the neuroglia the microscope revealed an unusual richness of its nuclei; at least bodies similar to these were abundantly strewn throughout the sections. Sometimes there was an apparent relative increase in the amount of neuroglia generally; occasionally, colloid bodies were found in the cortex, or pigment granulations, or microscopic patches which stained badly and had either a ground-glass-like or fibrous appearance. Not seldom were there various doubtful or equivocal appearances similar to some which are still matters of dispute between histologists.

The Blood-vessels of the Cortex.—Many vessels contained aggregations of blood-corpuscles, by which they sometimes were completely filled or were bulged.

Increase of the nuclei of the walls of the minute blood-vessels was a common appearance.

Sometimes molecular deposits or pigmentary deposits were seen in or upon their walls.

Either associated with these deposits or existing separately there were sometimes appearances of more or less irregular thickening or dilatation of the vascular wall.

Now and then some vessels had a soft molecular appearance; occasionally fusiform dilatation was seen; more rarely, capillary rupture and extravasation, so that vessels were surrounded by minute ecchymosis.

CEREBELLUM.—In the cerebellar cortex J. Luys described marked changes in the vessels and their contents even to solidification, hyperæmia, serous infiltration, and abnormal coloration; and throughout the cerebellum A. Voisin found hyperæmia, extravasation of blood-globules and of embryoplastic nuclei along the blood-vessels, in their walls, and even in the nervous tissue.

MEDULLA OBLONGATA.—Various changes, especially proliferation of embryo-plastic nuclei, have been described as extending between the cortex and the basal regions, but a higher interest naturally attaches to the lesions found in the medulla oblongata and its neighbourhood, inasmuch as hereabouts are situated the nuclear centres of those among the cerebral nerves whose functions first show impairment in general paralysis.

Magnan and others concluded the existence of ependymitis, and of diffuse interstitial peri-ependymary encephalitis, particularly in the fourth ventricle.

Here also Voisin often found an immense number of embryoplastic nuclei, or fusiform bodies, among the ganglionic nerve-cells of the nerve-nuclei and the fibres of the nerves. As in the brain, so here, they are most abundant in the walls of the vessels and in the perivascular spaces. "The cells of the nuclei of origin of the nerves in the medulla oblongata may themselves be altered and transformed into fat in various stages, and suffer a necrotic change" which is more advanced and extensive in the facial than in the hypoglossal nucleus, and still more than in that of the abducens. Also there may be marked fuscous degeneration of the cells of the nuclei of origin of nerves in the medulla oblongata, or atrophy of these nerves at their origin; with fatty degeneration of the glossal muscles and multiplication of the nuclei of their sarcolemma. In advanced cases fibrillary connective tissue is found in the vascular walls.

Bonnet and Poincaré describe the cells here as being occasionally fatty, and as always presenting rusty-hued granulations.

In one case J. P. Gray found the medulla oblongata of very small diameter, the pyramids small and indurated, the olivary bodies normal in appearance, and the cells of the corpora dentata well pronounced; the roots of the hypoglossus highly atrophied, its fibrillous structure barely marked; the ganglionic centre of the hypoglossus atrophied and in pigmentary degeneration; while there was only a slight pigmentary degeneration of the centres of the vagus and facialis nerves.

Connective tissue granulations of the ependyma, intense vascularization, and even minute hæmorrhages, and cellular proliferation, in the floor of the fourth ventricle, with destruction of the cells of the nuclei of origin of the facial and hypoglossal nerves are described by Clovis Gallopain. To the naked eye the softened, half-transparent, gelatiniform aspect of the grey matter is apparent: under the microscope are seen finely granular amorphous matter, isolated fatty granules, altered red blood-corpuscles, but especially rounded or oval cell-elements of homogeneous or slightly granular appearance. Moreover, the ganglionic nerve-cells of the hypoglossal and facial nuclei of origin appear as irregular masses composed of granulations, and often of reddish-yellow hue.

Cranial Nerves.—In these Voisin describes the following changes.

Olfactory nerves. Between the nerve-tubes effusion of blood-globules, and overgrowth of the normal nuclei, and collections of

hæmotosine here, or *in* the nerve-tubes ; around the vessels heaps of nuclei. In the olfactory bulb, sanguineous effusion in the walls of the vessels, and degeneration of the nerve-cells.

Optic nerve, third nerve, and sixth nerve. Overgrowth of their connective tissue, and compression of the nerve-tubes thereby ; and, in an advanced stage, granulo-fatty degeneration of the muscles supplied by the two latter.

The facial nerve is unchanged but the

Hypoglossal may be atrophied and present abundant embryoplastic nuclei.

THE SPINAL CORD.—The spinal cord, to whose grosser naked-eye changes in general paralysis Broussais, Boyd, Bucknill, and Joffe first more particularly directed attention, has latterly been subjected to much microscopical investigation.

In some general paralytics L. Clarke found softening of the spinal cord, "in certain parts to the consistence of cream." "In other instances, in which there was little or no external appearance of softening, I have found numerous areas of granular and fluid disintegration within and around the grey substance."

Besides changes in the spinal meninges C. Westphal described others in the spinal cord itself, either (1) in the posterior columns ; or (2) in the posterior sections of the lateral columns ; or (3) in these two tracts simultaneously.

(1). In the posterior columns he observed atrophy of the nerve elements, and growth of the connective tissue substance, which may be seen here and there in irregular plates varying in size and sometimes almost completely replacing the natural elements of the part. As a rule the alteration was most marked at the periphery of the column especially in the region of Goll's tracts, and the changes were often more extensive in the dorsal and lumbar than in the cervical region. Besides this, fat cells were described as sometimes being seen in fresh preparations, either free, or upon the walls of the vessels ; and, frequently, corpora amylacea ; and, occasionally, pale nucleated cell-elements.

(2). In the lateral columns. In fresh preparations, free nucleated cells were seen lying in the tissue ; and, in hardened preparations, nerve-fibres surrounded by a network of connective tissue with knotted points containing nucleolar structures. The appearances here mainly differed from those in the posterior columns by "the appearances and preservation of the reticular outline, the absence of the large irregular plates of connective tissue, and the constant presence of nucleated cells." When the lateral columns only were affected, the disease was confined to their posterior segments, and was usually most pronounced in the dorsal region.

The above change in the posterior columns (grey degeneration) affected also the posterior roots of the spinal nerves, and, as he thought, ceased at the fourth ventricle; while that in the lateral columns (chronic, or granular-cell myelitis) could be traced through the pyramids of the medulla oblongata, and the pons Varolii, to the foot of the cerebral peduncle, but no farther. That a continuation of the spinal cord changes have more recently been traced higher up will presently appear.

Now Westphal held that each kind of change was evidenced by special clinical features; the former occurring with a "tabic" form of gait, the latter with a "paralytic" form of gait.

Th. Simon of Hamburg denied the frequency or the characteristic nature of the "granule-cell myelitis" in general paralysis, or its intimate relation with the symptoms of the latter, inasmuch as he had not found these cells more frequent in the spinal cords of general paralytics than of others, not even when they are so abundant as to indicate a preceding myelitis. As well as in general paralysis the granule-cell myelitis was found in phthisis, chronic mania, and melancholia, and, especially, senile dementia. Simon also found a chronic spinal meningitis connected with phthisis.

The frequency of the granule-cells in the spinal cords of general paralytics, however, was admitted by Ludwig Meyer, but he rejected Westphal's view that they could be connected with the clinical symptoms, and, like Th. Simon, he found these cells in the cords of tubercular patients. He described fatty and calcareous changes in the vascular walls, and considered the point of origin of the granule-cells and fat granules in these, and other, cases to be the fatty degeneration of the walls of the vessels of grey and white substance, both in brain and cord.

With reference to the upward continuation of the changes in the medulla spinalis, Rabenau, who found the granular cells in the spinal cord, sometimes also found them continued upward through the medulla oblongata and pons Varolii into the crura cerebri, the radiating tract, and the centrum ovale of Vieussens. Both in the cord and in these parts did he also find degeneration of the blood-vessels.

Huguenin declared that the granule-cells may originate in general paralysis, from the nuclei of the neuroglial "cells," from those of the cells forming capillaries, or of the muscular coat of large vessels; from the connective tissue cells surrounding blood-vessels; from spindle cells of inner layer of cerebral grey cortex; and from the ganglionic nerve-cells. Huguenin, moreover, was of opinion that he sometimes traced the grey degenera-

tion of the posterior columns of the cord into the cerebral crura. Also that a rich deposit of cellular elements (emigrated lymph-corpuscles), which he could recognize in the internal capsule and corona radiata, bore some relation to the morbid process. In this view the "granular-cell myelitis" would follow the same course as the descending degeneration which succeeds to lesions of the radiations of the *basis cruris cerebri*, especially of the internal capsule.

The anterior roots of the cervical nerves were found in advanced fatty degeneration by Gray; who also observed increase of connective tissue; and accumulation of granule-cells in the post-lateral columns, and along the vessels.

Bonnet and Poincaré failed to observe sclerosis, or fatty degeneration,—or granulations of the fibres—in the cord. They found unimportant changes therein; such as rust-coloured pigment or granulations in the nerve-cells, close to the "ependyma." Occasionally, also, they found free droplets of fat in the stroma, or minute hæmorrhages. The ganglia on the posterior roots of the spinal nerves, and those interpolated on certain cranial nerves, may exhibit the same pigmentation of nerve-cells as in the cord; with, perhaps, black pigment or amyloid bodies in the stroma.

The principal changes reported by Ringrose Atkins in the medulla oblongata and spinal cord of certain general paralytics, and variously combined in different cases, were, fibrosis of vessels; pigment atrophy of the nuclei of origin of the eighth and ninth cranial nerves, and of cells in the anterior spinal cornua; atrophy of nerve fibres; coarseness and molecular degeneration of connective tissue, and general nuclear proliferation throughout; obliteration of central canal; and perivascular erosion with miliary sclerosis.

In the spinal cord, precisely as in the brain, Voisin found the primary lesion to be vascular and perivascular; followed by blastemic effusion in the nervous substance. Here also the vascular changes may advance to the formation of connective tissue in and around their walls. Both the lesions of the vessels and the effusion of embryoplastic nuclei in the nervous substance become more and more manifest as one nears, first the grey substance, and then the centre of the cord, the central artery being almost always extremely altered.

SYMPATHETIC GANGLIA.—Bonnet and Poincaré describe marked changes in the sympathetic ganglia, and especially in those of the cervical region, to the disease of which they assign the leading place in the production of general paralysis. They find a substitution of connective tissue and of adipose globules, for the

nerve-cells, in general paralysis; the connective tissue is greatly increased (sclerosis); heaps of adipose cellulos replace constellations of nerve-cells, and are sometimes tinted of a deep brown colour; the nerve-cells, now but sparsely seen, are pigmented; and pigment-masses may be scattered through the connective tissue and on the walls of the vessels. In the cervical ganglia the vessels may also be distended, and even varicose.

Thus, substitution of cellular tissue and of adipose "cellules," for the nerve-cells, is the essence of the change they describe.

SCIATIC NERVES.—On examining the sciatic nerve in some general paralytics Bevan Lewis found it diminished in size; and under the microscope there were seen;—a fasciculate atrophy of the nerve tubuli, involving both the medullary sheath and axis cylinder—defective staining of the axis cylinders—increased vascularization—and hyperplasia of the intra-funicular connective element.

CHAPTER VIII.

PATHOLOGY, AND PATHOLOGICAL PHYSIOLOGY.

PATHOLOGY.

(a). It is not within the scope of this work to speak with any fulness of the essential nature of general paralysis—either clinical or pathological.

As to the former—*the essential clinical nature*—one need only glance at the very different views that have been maintained on this question. That general paralysis is simply a complication, or even a termination, of insanity (doctrine of duality); or, on the other hand, that it is a distinct and special form of disease, evidenced by symptoms both mental, motor, and sensory (doctrine of unity); or, again, that the affection of motility is the primary and essential part of the malady, the mental affection, if present, being secondary and accidental; or, again, that there are two distinct kinds of general paralysis—one with, the other without, insanity;—are views each of which has had a distinguished following.

(b). Nor have the views as to the *seat* of the disease been less various; and some, combining in themselves the opinions of several, have declared that general paralysis arises from disease

of the spinal cord, or of the brain, or of the sympathetic nerves, or from a peripheral influence on the nervous apparatus, or without any appreciable lesion of the nervous system.

(c). *Cerebral Congestion* and inflammation. As to the much-vexed question of the essential nature of the pathological process affecting the nervous system in general paralysis, we may briefly note the important rôle so often assigned to cerebral congestion of an active kind. Of the several different views entertained on this subject it will suffice to state the following:—

That cerebral congestion is the proximate or determining cause of general paralysis.

That the repeated or persistent cerebral congestion causes irritation, and then a disorganizing inflammation.

That it acts by causing capillary obstruction, and consequent necrotic softening of the encephalon.

That the inflammatory cerebral congestion or the interstitial sub-phlegmasia is an epiphenomenon of degeneration.

That the cerebral congestion is secondary, and that the meningo-encephalitis, when it exists, occurs secondarily, and very late, and as a consequence of a failure of the general nutrition.

Here also our attention is claimed by a conflict of opinion as to whether the essential pathological change in general paralysis is principally and primarily of a more or less frank inflammatory nature, or principally and primarily of a degenerative nature.

Thus the observers of one group look upon an active inflammatory stage as being primary, those of the other look upon it either as secondary or as non-existent. To the minds of the former, again, the degeneration of cortico-meningeal vessels, of nerve-cells, or of neuroglia, is secondary; for the latter, these degenerations are primary.

1. *General Paralysis viewed principally and primarily as an Inflammation.*—In 1826 A. L. J. Bayle asserted that general paralysis “is the effect of irritation or inflammation of the cortical grey substance of the cerebrum, which directly disorders the functions of the brain. This irritation, or this inflammation, is in its turn the direct result of a chronic inflammation of the soft meninges, which commences at their interval or cerebral surface.” The chronic meningitis, he says, is sometimes accompanied by consecutive inflammation of the cortical grey substance of the hemispheres. In his view it was not that an acute inflammation of the meninges and brain had become chronic and produced insanity and paralysis—quite otherwise—for he stated that the lesion of general paralysis has a chronic course, is determined by its special proper causes, and never succeeds an acute inflamma-

tion. Very similar to these, also, were the views held by him in 1822 and in 1825.

In a single sentence, published in 1823, M. Georget refers symptoms like some of those in general paralysis to general chronic encephalitis. But he proceeds to confound this with mere diminution of brain-consistence in cachectic sane persons dying after lingering illness, such as phthisis. This, and his subsequent hostility to the views of Bayle, prove that the pathology and morbid histology of what is now called general paralysis were at that time quite unknown to Georget.

At nearly the same period L. F. Calmeil held that in general paralysis there was chronic inflammation of the encephalon which worked its principal ravages at the periphery of the gyri, in the grey substance, and in the envelopes of the brain; and that the chronic inflammation produced general paralysis by determining a special modification of the brain of unknown nature: and twenty years later Baillarger was of this same view as to the proximate cause of general paralysis. Similarly in 1859 Calmeil attributed the symptoms to chronic diffuse periencephalitis.

Deeming the specific morbid change in general paralysis to be a softening of the middle layer of the cortical substance of the cerebrum, Parchappe viewed the softening as due to an inflammatory process.

General paralysis is the result of chronic encephalitis said Belhomme. This encephalitis, at first superficial, and only producing slight symptoms, augments little by little, and invades the cerebral mass layer by layer, so to speak, until the central parts are reached and life is extinguished. In rapid cases, he said, the inflammation seizes on the whole encephalon. The cerebellum sometimes participates in the general inflammatory process.

The researches of Franz Meschede led him to conclude that general paralysis possessed as its pathological lesion a parenchymatous inflammation of the inner layer of the grey cortical substance of the cerebral hemispheres,—an inflammation affecting their nerve-cells principally.

Ludwig Meyer supported the inflammatory view: chronic meningitis plays an important part according to him, and chronic encephalitis generally follows. The inflammatory action starts from the walls of the capillary vessels of the grey cortex.

But before this Bouillaud had considered that general paralysis might be one of the terminations of general acute encephalitis. He observed that where patients do not succumb to a general acute encephalitis, and it, instead of ending in recovery, passes

into a chronic state, there slowly supervene mental alienation and "general paralysis." Previously he had attributed the symptoms of general paralysis to a chronic inflammation,—more especially of the cerebellum, and of the anterior part of the cerebral lobes.

One of his cases was also spoken of by L. Lunier as being due to acute hydrocephalus which had become chronic; and A. P. Requin observed several cases, which he believed to be general paralysis of the insane, to follow upon acute meningo-encephalitis or "cerebral fever." Yet, in the necropsy he records, the lesions were those of acute or subacute meningitis with fibrinous and purulent effusion, and, therefore, distinct from general paralysis, which he agrees is due to chronic diffuse encephalitis.

That the diffuse meningo-encephalitis in general paralysis might take on an acute form was maintained by A. J. Linas.

Dr. S. Wilks in 1866 inclined to the view that general paralysis is of the nature of a common inflammation, so that it bears the same relation to acute meningo-encephalitis or cerebritis as other chronic do to the corresponding acute affections—the same relation, he said, that a chronic rheumatic arthritis, affecting all the same structures, bears to an acute synovitis of a joint going on to involve all the tissues of the part; and he added that any form of chronic periencephalitis may be attended by the symptoms usually observed in general paralysis. Nevertheless, even in the same paper he called it a "peculiar inflammatory process." In 1868 he named the process "meningo-cerebritis chronica," or "peri-encephalo-meningitis." In his recently reprinted *Lectures* he has described the affection as "a disease of the cerebro-spinal centres having the same relation to acute inflammation as phthisis has to pneumonia, or a chronic rheumatic arthritis to an acute synovitis, or, more appropriately, a chronic Bright's disease to an acute nephritis."

V. Magnan concluded that there was generalized diffuse interstitial encephalitis, invading the brain both from the external surface and from the ventricular: while H. Schüle spoke of encephalo-myelitis chronica; complicated in the typical variety with a meningitis chronica.

It was believed by A. Lubimoff that the morbid process in general paralysis is a chronic interstitial inflammation. In the production of the phenomena of the disease he and others attributed an important share to the proliferation of the cellules of the interstitial tissue of the brain. After hyperæmias in the course of general paralysis the endosmotic conditions are changed, the neuroglia-cellules become swollen, and disorder the nutrition of the nerve-cells. Finally, by their frequency and repetition the

hyperæmias alter the nerve-cells essentially, and irreparably impair the psychical functions.

Hitzig agrees "that general paralysis in a more restrained sense, and so far as concerns the brain, is a chronic or sometimes, rather, a subacute interstitial (peri) encephalitis, which in course of time leads to destruction of the ganglion cells, and to atrophy of the brain."

That the morbid change is disease, approaching the nature of chronic inflammation, of the outer layers of the grey substance of the brain convolutions, was the view of T. S. Clouston.

Aug. Voisin also adheres to the inflammatory view, certainly in 1875, 1877, and, again, in 1879: and inflammation of the periphery of the encephalon was also deemed by Burlureaux the essential lesion in general paralysis: while Dr. Crichton Browne has joined himself to those who consider general paralysis to be of inflammatory nature.

Well placed from the first by the weight of so great authority as that of Bayle and Calmeil, the doctrine of the inflammatory nature of general paralysis for many years ruled supreme. Not that the precise character of the inflammation or the elements primarily affected by it were agreed upon. As to what part of the encephalon or meninges was the special seat of inflammation, as to whether the latter was interstitial or parenchymatous, and as to its relations with ordinary acute inflammation of the same structures, opinion was divided, and discrepancy rife.

In fact, at an early period departures were made from the pure and simple doctrine of inflammation, and the rôle assigned to the latter was by no means always the same.

It has been seen that almost at the outset Calmeil spoke of a special modification of the brain, of unknown nature, as being produced by the chronic inflammation, and as, in its turn, conditioning the symptoms; and following him, no doubt, Daveau gave expression to similar views.

Baillarger described two orders of lesions as characterizing different cases in general paralysis. I, The inflammatory—those due to peripheral meningo-encephalitis: and, II, those dependent upon hydrocephalic or serous effusion, with atrophy or softening of brain. Lesions of this second order would usually be distinct from those of the first, and from the works of Dance, Moulin and Rochoux have been selected cases of general paralysis following chronic hydrocephalus, or serous effusion consecutive to apoplexy. This latter view, I think, must be rejected: indeed Griesinger gave the weight of his name to the statement that apoplexy is never the foundation of general paralysis.

Then, again, an identity was assumed by Lunier between general paralysis of the insane, hydrocephalus of adults and of the aged, and the chronic affections of meninges or of encephalon which succeed the acute inflammations of these structures. He inclined to the doctrine of Broussais, which would assign the changes in general paralysis to "irritation," and held that the first step was cerebral congestion, or, more rarely, intra-cranial hæmorrhage.

Views such as these may be deemed as in some sort a compromise, or transitional, between the exclusively inflammatory doctrine and the views immediately to be mentioned.

II. *General Paralysis viewed as primarily (and principally) a Degeneration.*

L. V. Marcé questioned the inflammatory origin of general paralysis, and thought it due, rather, to a chronic congestion with exudation—a view to which even Calmeil inclined for the milder simple cases. And it has been held by some that the disease is essentially degenerative, that, for example, a degeneration of the vascular walls occurs in the pia-mater and cerebral cortex, which gives origin to obstruction and derangement of circulation; and these to derangement of nutrition, while there is secondarily a spreading and devastating formation of connective tissue in the cortex and elsewhere, leading to destruction of the nerve-elements. Delasiauve suggested that there was in general paralysis a degenerative element, of which the sanguineous stasis, less active than passive, was the result and not the cause. These are very different from the older view that general paralysis is due to an inflammation of the brain and meninges, from irritation brought on by repeated active congestions.

Bucknill spoke of general paralysis as being "essentially a disease of nutrition affecting the whole nervous system." "It consists in some vice of nutrition whose nature is yet unknown, but whose extent embraces the whole of the nervous system, and is by no means limited to the encephalic centres." Again, "The morbid processes in the brain are not encephalitic."

Much to the point is the summary given by the Swedish physician Salomon who, after referring to degeneration of the vascular walls and of the neuroglia in general paralysis, asserted that this disease "consists essentially in a degenerative process in the adventitious membrane of the vessels of the pia-mater, and in the tissue connecting the elements of the cortical substance of the brain (neuroglia), which degenerative process in its development causes the change of the grey cerebral cells into an inert mass." Something more than hyperæmia is required to effect this; and

that additional element is supposed to constitute the *peculiarity* of the disease, and to be of a degenerative nature.

More recently (1868 and 1875) Bonnet and Poincaré consider that inflammation is not necessarily present in general paralysis, and that when it does occur it is late in the course of the disease, and is the result of defective nutrition. They agree with the earlier writers as to the existence of cerebral congestion, and the *rôle* that it plays; but not as to the *primum movens* of that congestion. Especially do they agree with Bayle as to the unity of the disease and the congestive element; while they differ from him, essentially, as to the interpretation of the symptoms and the point of departure. They believe that the alterations found in the encephalon are the consequences of disorders of the cerebral circulation following impairment of the function of the sympathetic ganglia, owing to their diseased condition, as described in a preceding chapter. They say that in these ganglia they find the anatomical point of departure of the disease; and that the changes in the encephalon are only the consequences of disorders produced in the cerebral circulation by the sclerosis and fatty substitution in the cervical sympathetic ganglia,—changes which have a paralyzing effect upon the vessels. "All the alterations we have described bring on disorders of nutrition in most of the organs—disorders which tend to fatty degeneration, or other modifications of their elements, and betray themselves on the physiological side, at first by ataxy, and then by prostration of the functions both of the life of relation and the vegetative." Thus, in their view, vaso-motor lesions and disorders are of the very essence of the disease.

Dr. G. F. Blandford inclined to the views of Bonnet and Poincaré as published by them in 1868.

Very recently J. Luys joins himself to those who believe that the morbid process in general paralysis is a true, diffuse, interstitial sclerosis of the neuroglia of the nervous centres. In his view this acts both directly on the true nerve-elements, and indirectly by implicating the vessels. It starts from different points in different cases, sometimes in the white substance, sometimes in the cortex, sometimes in the spinal cord, and hence its forms are very different at the commencement in different cases. Of course, however, it may be argued that this sclerosis is inflammatory in nature.

There is much to commend a former statement by Luys, who, after ascribing the changes of general paralysis in and about the vascular walls and perivascular spaces seated in the nervous centres, to the effects of congestion, even if of a passive kind—

and showing that the process is a complex one, in which both the over-supply of blood and the proliferative reaction of the vascular walls play simultaneous and important parts,—proceeds to show that this is a special form of plastic congestion; intermediate to simple congestion, whose barriers it has overleaped; and to exudative congestion, unto which it has not attained;—it is more than mere passive congestion by reason of its exudative tendencies, yet it is not inflammation, at the outset (at least), by reason of the absence of primordial erethism and of purulent formation.

To the preceding we may add one or two points of pathological interest.

(a). General paralysis has been considered by some as primarily and essentially a disease of the spinal cord, and others have given a most prominent place to the spinal lesions [*vide morbid anatomy supra*].

(3). The view was formulated by Ach. Foville, that although in the vast majority of cases general paralysis is an independent primary cerebral affection, yet that, exceptionally, it is consecutive to some other affection of the nervous system, and results from the propagation to the cerebral hemispheres of disease either from the spinal cord,* or from a cranial nerve.†

As to locomotor ataxy, in this relation, cases have been published by Simon, Vulpian, Liouville, and perhaps the second and third cases by P. Nicol. Westphal's cases may also be compared with these.

Westphal has also observed general paralysis follow atrophy of the fifth cranial nerve and Casserion ganglion; and, also, atrophy of the olfactory nerves. Falret's example of this "propagation" was one from disease causing partial palsy of the third cranial nerve. Three of his cases, Nos. 89, 90, and 91, were claimed by Mobèche as belonging to the same category as those of Foville: even for a long period had amaurosis preceded the symptoms of general paralysis in them. General paralysis has been observed by Baillarger consecutively to progressive muscular atrophy.

(γ). The homology between the morbid changes in general paralysis and in certain forms of Bright's disease was noted by Salomon in 1861; and the toxic origin of general paralysis (in some cases at least) was long ago suggested by Dr. Jno. Hitchman; and more recently by Burman, inasmuch as the cerebral changes bore a strong resemblance to those of cirrhotic kidneys

* As that producing, *ex gra.*, locomotor ataxy.

† As that producing, *ex gra.*, amaurosis, ptosis, strabismus.

and livers, notoriously linked to toxæmia, especially of alcoholic origin. A toxic origin has also recently been suggested by I. Ashe, but there seems to be little support for his hypothesis that general paralysis may be due to retention of an undue amount of phosphorus in the system; that it is a form of chronic phosphorus poisoning, in fact.

(δ). As bearing upon certain pathological changes in general paralysis it is well to bear in mind how, in active states of the cerebral circulation, certain natural obstacles to the local circulatory freedom bring about chronic thickening of the pia-arachnoid, increase in size of Pacchionian bodies, increase of subarachnoid fluid, thickening of the skull inwards, and some increase in the firmness of the brain. (Wilks and Moxon. *Path. Anat.*, 2nd Ed. p. 205.)

(ε). The arachnoid cysts sometimes found in general paralytics result, either from the transformation of blood effused into the arachnoid cavity, whether by primary hæmorrhagic extravasation, or, probably, by rupture of fattily degenerated vessels of pachymeningitic neo-membranes; or perhaps, occasionally, from fibrinous effusion.

THE PATHOLOGICAL PHYSIOLOGY OF GENERAL PARALYSIS.—To trace the many symptoms of general paralysis to the various effects upon physiological function of the several stages of the morbid process, or to its several kinds, or to the several localities mainly affected, is a task which has varied in its execution with each advance, or supposed advance, or each caprice of fashion, in physiological and in pathological teaching.

Broadly speaking, the motor signs and the mental symptoms more usually proceed in somewhat parallel lines and with somewhat equal paces, and in reference to this association Maudsley has written that "not only does the morbid state of the motor centre lead to a difficulty of expression by the appropriate movements, but the diseased motor intuition enters into the intellectual life, and in conjunction with morbid ideas there, gives rise to all sorts of extravagant and outrageous delusions as to personal power." Again, he points out how more than usually impossible is the correction of the delusions, on account of the failure of the muscular sense partially closing the avenue whereby a knowledge of the qualities of objects is acquired, and leaving the patient a prey to internal disorder.

In the first two of the three stages he described, the "paralytic" symptoms were attributed by Bayle to the effects of congestion of the pia-mater (and brain), and of thickening of the meninges; and in the third and last stage, partly to the same

congestion, but especially to the pressure exerted by the now excessive arachnoid, subarachnoid, and ventricular serosity.

The delirium, exaltation, agitation, and furor, he assigned to the irritation exercised by the inflamed meninges on the cortical substance of the convexity and internal surface of the cerebral hemispheres, and thence on the entire encephalon:—the ambitious ideas to the indirect effect on the cortical substance, and successively, on the whole brain, of the sanguineous congestion of the pia-mater and of the inflammation of the internal face of the arachnoid:—the dementia to compression of the brain by serosity:—the apoplectiform attacks to sudden sanguineous congestion in the vessels of the pia-mater and brain:—and the convulsions to consecutive inflammation of the grey cortex of the cerebrum; or, rarely, to sudden serous effusion upon its surface, or into its ventricles.

Among the earlier observers were some who attributed the mental symptoms, in general paralysis, to the diseased state of the cerebral cortex, and the motor to that of the cerebral medullary substance.

Dagonet ascribed the general motor paresis to bulbar lesion, perhaps due to extension of the morbid process from the brain-cortex.

Clouston found the anterior and upper convolutions of the brain to be the parts most affected in the cases where the mental symptoms had been most severe,—the base and fourth ventricle where the epileptiform convulsions had been prominent,—and, perhaps, Broca's convolution where the speech had been specially affected.

When the articulation was much affected Spitzka found the lesions specially about the insula, the operculum, and the upper aspect of the temporal lobe; as well as the lenticular nucleus (the part, he says, with greatest vascular lesion in general paralysis). When the paralysis of the extremities was very marked the lesions were most advanced in the upper parts of the præ-central and post-central, and contiguous, gyri. With slight motor paresis, and marked mnemonic failure, were lesions of the second and third frontal gyri.

To pass more particularly to individual signs and symptoms.

Speech. Bouillaud viewed the cerebellum as containing the co-ordinating centres for the movements of the lower extremities;—the anterior cerebral lobes as the co-ordinating centres of speech.

He ascribed the stammering, or loss of speech, in general paralysis to chronic inflammation of the anterior part of the cerebral lobes, and to a similar alteration of the cerebellum he assigned

the inco-ordination of the lower limbs, the failure in locomotion and in maintaining the equilibrium. For him there was no paralysis here; but a "déséquilibre" of movement as, said he, in an animal deprived of the cerebellum. And he has recently confirmed the same view as to the speech disorders of cerebral diseases.

That a relation subsists between the speech-affection and the mental condition ("dispositions d'esprit") of the patient was a belief supported by Lasègue, or at least, like others, he denied that the incertitude and hesitation of speech were proportional to the trembling of the tongue or to the vermicular movement of the lips: and Linas considered loss or, rather, impairment of the memory of words (Bouillaud) an element in the speech-disorder of general paralytics, of which the other elements were the so-called "paralyses" of the tongue, lips, and face.

The lesions of certain nerve-nuclei beneath the floor of the fourth ventricle were indicated by Clovis Gallopain as the anatomical basis of the disorder of speech in general paralysis; and like Marcé he considered the dysphasia as dependent upon, and directly proportional to, the labial and lingual ataxy. In stating that the difficulty of articulation resulted from trembling and irregular quasi-convulsive contractions of the muscles of the tongue, lips, face, and sometimes lower jaw, Marcé, even if not entirely right, was in advance of the older writers who attributed the articulatory disorder to *paralysis of the tongue*.

On the other hand, the disorders of speech in general paralysis were several years ago placed by M. Voisin in three groups, each of which he assigned to certain lesions occupying certain sites. The early hesitation and slowness of speech, and the omission of words are mainly of psychical origin, and due to lesions of the frontal lobes and insula, to which lesions of the fibres from these parts to the bulb may add themselves. Secondly, the trembling, stammering, and jabbering are somatic in origin, are ataxic, and due to bulbar lesions. Finally, the mutism is due either to absolute failure of ideas; or to advanced degeneration of the bulbar nerve-nuclei, or of the lingual muscles;—or to lesions of the gylar speech-centre, of the fibres thence to the bulb, and of the corpus striatum.

Other Motor, and especially Ataxic, Symptoms.—Voisin also thinks that the trembling and twitching of the lips, face, and tongue are due to lesion of the roots, and of the cells of the nuclei of origin, of the facial and hypoglossal nerves in the medulla oblongata;—that the ataxic disorders of the limbs are due to lesion of the posterior part of the spinal cord or of the

posterior spinal meninges;—that the pupillary inequality, sluggishness, and dilatation are due to lesions of the origin of one or both of the third cranial nerves; and that pupillary contraction is due to lesion of the roots of first and second dorsal pairs of nerves, or of the cilio-spinal centre of the cord situate between the fifth cervical and the third dorsal; or, later, that the pupillary changes are often due to vaso-motor spasm or paralysis, or to lesions of the optic thalami;—that the temporary pareses and paralysees are due to temporary hyperæmiæ; but if persistent are due to a complication, such as cerebral hæmorrhage, localized meningo-encephalitis, or unilateral cerebral atrophy of the side opposite to the hemiplegia.

Convulsions.—At first attributed to secondary inflammation with softening, and occasionally, meningeal adhesion of the cerebral cortex; or, rarely, to rapid serous effusion, the convulsions of general paralysis were at a later period attributed to seizures of cerebral congestion,—still later, to sudden anæmia from contraction of cerebral blood-vessels induced by various “irritations,” and probably connected with an increased reflex-excitability of the medulla oblongata; and, later still, to any one of several causes such as meningeal ecchymosis, pachymeningitic fluxion, intense cerebral congestion, or even minute hæmorrhages from rupture of ampullary dilatations of arterioles;—while by one of the most recent observers, Oraggi, they are ascribed to stasis in the meningeal veins, easily brought about by disturbances of the circulation when the meninges are thickened.

In the *Journal of Mental Science* for January, 1876, I have attempted to trace a relation between the special distribution and range of the convulsive symptoms, and the speech-troubles in general paralysis, on the one hand, and on the other, the distribution of the cortical changes, especially of that associated with adhesion to the meninges. In this, as well as in every case examined for years before that period and since, I have kept minute records of all the cerebro-meningeal adhesions, in order to test recent views as to the localization of cerebral functions, by comparing the differences in the distribution of the adhesions with the differences observed in the clinical symptoms, in different cases. A large number of these records as to adhesions are summarized in my paper in the *Journal of Mental Science* for April, 1878.*

That which I wished to test Dr. Ach. Foville has since assumed, and the motor paresis and the spasmodic disorders of different parts are consigned by him, each to lesions of special cortical

* See the Second Part of this work.

centres. Assuming that the cortical lesion in the fronto-parietal convolutions is constant, he believes that the progressive disorders of the motor functions in general paralysis are explained;—at first, by the excitation produced in the different motor centres situated in these convolutions, and due to the hyperæmia—then, by the successive congestive attacks;—and, finally, by the progressive sclerosis of the period of decline. These views are purely hypothetical, but of course are those naturally flowing out of recent researches on the localization of cerebral function.

Similar views, carried further and applied to the explanation of the mental and sensory symptoms, have since then been published by Dr. Crichton Browne, and he has related the histories of the patients whose brains are figured in the plates with and by which he has enriched his paper and exemplified the distribution of the adhesions.

More lately, Dufour has traced out the adhesions and atrophies in several cases of general paralysis, and endeavoured therefrom to explain the functional localizations in various forms of the disease.

On the other hand, writing before the more modern experiments had been made, Dr. J. Luys considered the various motor disorders in general paralysis to be expressive of a profound and progressive derangement in the *cerebellar* function. For the progressive general muscular enfeeblement and the local motor perturbations producing disorder of speech, and of gait, paralyzes, convulsions, and so forth, are also observed in different cases of cerebellar disease, and are general or partial according to the amount of cerebellar involvement. To this there is an exact similitude, he says, in general paralysis, save for the tendency to generalization of the morbid process in the latter. Yet the unequal invasion in different cases leads to a further specialization of the symptoms. In this view the inessential nature of mental symptoms in general paralysis is obvious.

Psychical Symptoms.—As to the production of the psychical disorders in general paralysis several views have been expressed above. To them we may add that Meynert, and before him Bouilland, attributed the loss of memory in general paralysis to lesion of the frontal lobes.

The failure of attention, of intelligence, and of the power of judgment in general paralysis are ascribed by Crichton Browne to lesions of the frontal lobes, and the exalted delusions to morbid stimulation of the so-called higher, and cortical, motor centres in the posterior frontal and in the parietal regions.

The rapid flight of ideas in general paralysis is attributed by Spitzka to hyperæmia of the cerebral capillaries, the incoherence

to patches of the hyperæmic engorgement passing over into stasis:—attacks of stupor to more extreme and general stasis:—and subsequent lucidity to the breaking up of the opalescent hyaline cylinder into which the blood-corpuscles had coalesced during the stasis.

Luys thought the cerebeller innervation exercises a mediate stimulating influence upon the cerebral activity. Roused to over-action by the morbid process, the cerebellar innervation is projected with greater vehemence into the corpora striata in the early stages;—but this gives place finally, to exhaustion and diminished influx. At first the increased cerebellar influx leads to grand ideas in general paralysis; but, finally, a sort of asthenia strikes the psychical functions when the cerebellar influence becomes attenuated, the coexistent feelings of prostration, melancholia, and hypochondria, betraying a sort of unconscious appreciation of the progressive destruction of foci of cerebellar innervation. The *cerebral* cortex, therefore, acts automatically, according to its habitude, but now upon fictitious data transmitted to it from the cells of the corpora striata—in a state of erethism, or the reverse. Thus it becomes the sport of true hallucinations, of which sometimes hypersthenia, sometimes asthenia, is the common basis.

I would utterly reject this fanciful theory.

Confronted with these various, and often irreconcilable, views one may proceed to examine the subject anew.

Proposed View.—In the interpretation of the various symptoms of general paralysis, it may be broadly stated that the morbid process in the nervous system first deranges, and then destroys, or tends to destroy, the functions of the parts affected.

Yet is the interpretation surrounded by grave difficulties. For:—1, The morbid process is modified in different cases.—2, Its extent varies likewise.—3, The part or parts at which it begins, also vary in different instances.—4, In some cases the greater portion of the cerebro-spinal system may be more or less involved in a general disturbance of nutrition and disorder of function, while, in others, the diseased action may long be *comparatively* localized.—5, As different nervous districts are successively and progressively implicated, it usually happens that the function of one part is merely exaggerated or disordered at the same time as that of another is practically destroyed.—6, The destruction of an inhibitory centre, under these circumstances, will permit relative over-action of functionally related centres, which have escaped from its regulative influence.

Motor and mental nervous centres are markedly implicated in

the morbid process. The motor disorders cannot be fully explained by any secondary disease of the fibres which merely transmit motor impulses, although this disease plays its share in the later stages.

On the one hand, as the principal lesions of general paralysis apparently are situated in the cerebral cortex, and, on the other, as recent investigations tend to show that in the cortex are situated not only the mental centres, as ordinarily believed, but the highest motor centres also, hence there has arisen an inclination to adopt a well-knit theory that the mental and motor symptoms in general paralysis are due to a widely-spread cortical lesion, including within its area of action both the motor and the mental centres, to which indeed we may add the sensory.

And here I would bring to the notice of modern experimenters that practically this physiological view as to the existence of cortical psycho-motor centres is not so new as they deem it to be. It is constantly implied by Bayle. Parchappe, again, speaks of softening of the cerebral cortex in general paralysis as producing the diminution of the muscular forces, and difficulty in the movements co-ordinated by the will. On this point also may be consulted the *Revue Médicale* for Sept. 1846, as to lesions of superficial parts of the brain producing paralysis; and many cases in Lallemand's works.

As to the particular parts of the cortex necessarily involved, under this theory, they vary as one adopts the conclusions of this or that physiologist. But, watching the proceedings of the experimentalists, one may utilize the actual outcome of their interference with the brains of lower animals, and yet be animated by a feeling of caution and reserve in accepting many of the rigid conclusions drawn therefrom.

If the true higher motor centres are not *in* the cerebral cortex they are at least in evident intimate functional connection therewith—a functional connection which undoubtedly involves a pathological alliance—a community in pathological suffering—a true sympathy—also.

Be the functional relation what it may between centres in the cerebral cortex on the one hand, and basal and spinal motor centres on the other, it must not be forgotten that in a certain group of general paralytics the first apparent symptoms are spinal, and that these are soon followed by mental symptoms; apparently from subsequent invasion of the cerebral cortex.

Believing that almost invariably the disease is primarily and mainly of the cerebral cortex and its tunics, I prefer to continue minute observations of the cadaver, comparing them with the

results of physiological experiments, rather than to be entirely lulled by the apparent facility of any new, and as yet unproven, doctrine. Guided by that scheme of localization (Ferrier's) which is best known in this country one would be led to assign the *mental* disorder and failure in general paralysis to the morbid process or change, of the greater part of the cortex of the frontal lobes—morbid stimulation of the motor and sensory centres also modifying the mental condition and conducing to mental disorder :—the *motor* troubles mainly to that of the central and paracentral districts ;—the *sensory* affections to that of the temporo-sphenoidal, and part of the parietal, lobe ; and any perversion or destruction of *organic feeling*, to that of the occipital.

Restricting one's attention to the clinical aspects and to the morbid histology as actually observed, the course of general paralysis is probably somewhat as follows.

In the vast majority of cases the cerebral cortex is primarily affected, the meninges usually being more or less involved almost simultaneously.

In many cases the morbid process apparently is most active, and at first active only, in circumscribed regions of the cerebral cortex. In others the morbid action is more diffused.

Taking the mass of cases the convolutions of the frontal and of the parietal lobes suffer more than those of other parts of the brain.

The pia-matral adhesions to the cortex, already described, often form a most valuable index to the localities principally diseased in general paralysis. These adhesions vary very much in their extent, degree, and situation, in various cases, in correspondence with the greater activity of the morbid process in different circumscribed regions of the cortex, as just mentioned.

The morbid process in general paralysis is primarily set up by excessive, irregular, protracted activity and over-strain of a larger or smaller number of the active functioning elements of the cerebral cortex, which subserve the higher faculties of the organism. It is usually admitted that these active functioning elements are the so-called ganglionic nerve-cells of the cortex. Of the most potent and frequent causes of general paralysis each, in its own way, brings about the primary step to which we refer, namely, the excessive, irregular, protracted, activity or over-strain of a larger or smaller number of the nerve-cells.

Partly in consequence of the different modes of action of the several causes, partly owing to the varieties in habits, work, and circumstances of the patients, producing habitual relative over-activity, activity, or disuse, of this or that part of the brain, and,

hence, relative higher development in the one case, and relative feebleness of action and simplicity of inter-communication in the other;—and partly owing to hereditary and to diathetic causes and, possibly, temperament as well;—it comes about that the cortical regions or centres most severely affected vary, as we have said, in different cases.

Over-activity of this kind and over-strain induce contemporaneous hyperæmia of the part, and this hyperæmia tends to keep in action its own causes. From frequent repetition of this condition the normal tonus of the arterioles is gradually lost, and not only in the cortex but in the overlying meninges also. Hereby is prepared the way for sudden or protracted meningeal and cerebral hyperæmias, which embarrass the brain-circulation and brain-nutrition in a more or less protracted manner, and leave behind them more or less permanent effects or traces.

It may be that the exhaustion from over-excitation of the cerebral nerve-cells induces vaso-constrictor paralysis:—*i.e.* paralysis, ever-renewed, of the vaso-constrictor filaments, usually assigned to an origin in the cerebro-spinal system through the sympathetic. But it is by no means clear that the vascular dilatation and sanguineous over-fulness may not be kept up by frequent, or ever-repeated, direct, or reflected irritation of the vaso-dilator or vaso-inhibitory filaments supplied to the local peri-vascular vaso-motor centres;—centres first indicated by Goltz—centres which maintain the normal arteriolar tonus—centres whose stimulation direct, or propagated, causes contraction of the blood-vessels animated thereby; and the inhibition of which causes vascular dilatation; the inhibiting influence being transmitted to the peri-vascular ganglia by the vaso-dilator or vaso-inhibitory filaments. Yet paralysis of vaso-constrictor fibres would promote that vascular dilatation after nerve-section, which Goltz would limit to irritation of vaso-dilator fibres.

Furthermore, we know that vaso-motor centres are situate in the cerebral-cortex, as well as in other parts of the nervous system, and that the irritation or destruction of these may influence the blood-supply, of the brain, of other parts, and glandular secretion. That is if we accept the conclusions of Brown-Séquard, Eulerberg, Landois, and others.

But be this as it may, we resume the consideration of the repeated, and more or less persistent, cerebro-meningeal hyperæmia. In consequence of this there is distension of the vessels, circulatory impediment, irritative overgrowth of the connective nuclei of the walls of the vessels, and probably also of the

neuroglia, while others of the nuclei and cellules often termed embryoplastic, or their materials, are, perhaps, directly effused. Other changes occur in both, also, as a result of the lowered standard of the local processes of nutrition. Moreover, out-wandering of white blood-corpuscles, and escape or extravasation of red blood-corpuscles, may further choke the parts. There is a constant tendency to diffusion of all the morbid processes, and among the macroscopic changes are a thickening, opacity, and œdema of the superjacent meninges.

But in the meanwhile the nerve-cells of the parts diseased, under the influence of morbid and excessive activity, have failed in their nutrition, while *they* also feel the effects of the surrounding vascular and neuroglial changes ;—hence their swelling, cloudiness, and final degeneration. Then, if a chronic, mild, adhesive form of inflammation sets in, fibrinous effusion, and the subsequent production of connective tissue assist in more completely involving the nerve-cells, and in tying down the membranes to the cortex.

These changes proceed in the usual degenerative course, and finally as a result of them we find the processes of the nerve-cells cut off by the way—the cells themselves atrophic and degenerate—the blood-vessels fatty, calcareous, pigmented, and misshapen,—and the, formerly hyperplastic, neuroglia now atrophied.

The diseased process having once set in it must produce a series of effects in any given centre or region. Let us see for a moment what occurs in such centre or region, (1) when considered theoretically as if detached, and (2) when the total effects of the lesions of all the centres diseased are considered.

In the simplest case of all there is merely progressive impairment of the function of certain mental, motor, and sensory centres,—any over-action observed being merely the result of the partial withdrawal of the normal inhibitory influence of higher centres, and hence freed action of lower centres. In these cases hyperæmia is either less, or is negatived in some of its effects by the predominance of parenchymatous, or of interstitial, changes, or of both ; which, as it were, jugulate at its very birth the functional energizing of the active elements.

But in the majority of cases there is more or less over-activity of an irregular and inharmonious kind. The nerve-cells, embarrassed by the surrounding neuroglial and vascular changes, and flooded with superabundance of blood, discharge fitfully and irregularly. Their activity, excessive in amount, is irregular in rhythm and inharmoniously adjusted, or inco-ordinate, as to normal functional relationships.

A. Mental Centres.

(a.) From disorder of this kind affecting the centres which are concerned with the anatomical substrata of purely mental function there is embarrassment in mental operation—inability for prolonged definite effort directed to any distinct purpose—inattention—a want of fixity of thought and of determination—a return to childish ways, and childish and more simple modes of thinking—a forgetfulness—and a failure to register and retain impressions—a dimming of the lustre of the higher virtues, of the general moral and æsthetic culture.

Still later, incoherence and feebleness of thought, and a disappearance of all sense of shame, of all moral feeling, are observed:—while mental confusion, and failure of memory, of perception, and of judgment are painfully obvious. All these psychical troubles may be assigned to the morbid process in the frontal lobes of the cerebrum.

(β.) But mingled with the above in the early stages there is often *Exaltation or ambitious delirium* in some of its phases. Partly does this occur as a result of the unwonted and morbid stimulation of the ideational centres by hyperæmia, which rouses old paths of ratiocination, and brings vividly into consciousness the old day-dreams, the relics of ambitious thought and of long and fondly cherished visions begotten of the mirage of hope—the whole taking a gay colouring, either from the excitation of the brain generally, or of definite parts of it possibly connected with the representation of the organic and other feelings. Partly from the above, then, comes the ambitious monomania or expansive delirium, and partly as a result of the morbid excitation of cortical motor centres giving rise to the subjective impression of an enormous outflow of energy—thence to a pleasurable feeling of power and force, and thence, mayhaps, extending beyond its primary sphere to tincture all the thoughts, and to swell the happy patient with exuberant spirits, with exultation, and with extravagant notions as to everything in relation with himself, his honours, wealth, position—extravagant notions which the failure of perception and of the intellect generally no longer permits him to doubt, much less to appreciate in their true character. The hallucinations and illusions,—especially perhaps illusions of the muscular sense,—from which the patients may suffer can also originate or foment their expansive state of feeling and idea.

Again, what with hyperæmia, protracted excitation, and the *relative* embarrassment of the highest centres, there may be excessive and discordant uncontrolled activity of the lower; and, in consequence, protracted or paroxysmal mental excitement.

(γ.) But what as to the cases in which *hypochondriacal or melancholic symptoms* are found, either sharing the throne with ambitious delirium, or excluding it temporarily, or, more rarely, excluding it permanently? They are rare as prodromes in my experience. When present they more usually occur in the middle and later stages. If the early activity of the disease-process, with its hyperæmia, leads to gaiety of feeling and optimistic delusions there would be little wonder if, after its force was largely expended, the centres of motion and sensation, now exhausted and partially destroyed, gave origin to organic feelings mutilated and modified, to subtle impressions of constraint, limitation of power, and prostration of energy,—thence to depressing emotion, and thence to ideas of the hypochondriacal or melancholic order. Yet a recrudescence of the primary morbid process in the same centres, or its activity elsewhere, may traverse these secondary effects and restore the exaltation and grandiose ideas. Not chimerical is this view that hypochondriacal delirium may be due to lesions of the cerebral cortex; the various visceral organs as well as the musculature are represented therein; by its experimental local irritation or destruction are these viscera affected; in the brain-cortex do organic sensations, as well as special sensations, find intimate alliance and highest representation. The early and rapid—or late and slow—destruction of motor centres, also, is adapted to call forth hypochondriacal or melancholic conceptions, the very antitheses of those former grandiose ideas the outcome of the wild whirling tide of life, in the motor ganglionic cells, begotten of hyperæmia.

Again, it is probable that temporary vaso-motor disturbance in certain regions of the encephalon may not unfrequently bring about a temporary mental change, and hence a transitory hypochondria or melancholia.

So, too, may alterations in the constitution of the blood account for certain of the mental phases, as the hypochondriacal or melancholic. In relation to this we may bear in mind the mental colouring usually associated with certain states of toxæmia; as, for example, the depression attending cholæmia; the ill-temper, anxiety, and depression of chronic lithæmia; and the apathy and unconcern of pyæmia. So also in phthisis; bright, swift, and lively as are often the mental powers in the early periods, yet later,—and when the blood is probably much altered,—caprice, fickleness, variability, and impatience are observed far more often than the so-called *spes phthisica*.

Again, the mental depression may be merely an ultimate reflex effect of impressions starting from some distant organ. The hypo-

chondria of general paralysis may, like simple hypochondria, have its rootlets in some visceral or some peripheral affection or organic disease; in some disorder or loss of sensorial function; whether of the special, as evinced by hallucinations, illusions, or failures or losses of the special senses; or in failure or perversion of common, or organic or visceral sensation, as suggested by Maudsley; or in various neuralgiæ, hyperæsthesiæ, and anæsthesiæ, or in spasm of tubular or hollow organs, or of voluntary muscles. Now these may be present from the outset, and the case may take the melancholic or hypochondriacal form from the outset, also; or they may occur late in the course of general paralysis, and produce an intercurrent and temporary hypochondria. These patients are inapt to obtrude their sufferings.

Vivid hallucinations of a distressing nature may call forth melancholic conceptions; and when general paralysis has arisen from depressing mental causes it may put on the melancholic form. Or when the patient finds that his imaginary untold wealth and power avail him nought *quoad* his actual possessions, liberty, and enjoyments, he oftentimes, in the early stages, becomes wrathful, or weeps and sobs like a grief-stricken child.

Finally, when melancholic symptoms in general paralysis are more permanent, or when throughout they are the most striking mental features, next to the failure of mental powers, I would refer to the evidence given below that in many cases the phenomena, perhaps, owe their explanation to the predominance of the morbid changes in the left cerebral hemisphere as compared with the right.

B. Motor Centres.—If the centre under consideration should be a motor centre, besides the motor intuitions and the motor element in ideas involved, as already described, there will be the production of certain motor symptoms. But here the cortex is by no means alone involved. The vast disease at the periphery of the brain may be followed by secondary disease travelling downwards through the white substance to the basal ganglia, and thence downwards; often to break forth, so to speak, with renewed energy in the medulla oblongata and spinal cord, and in the nerves thence derived. Nor, from this point of view, should the experiments of Lussana and Lemoigne be left unnoticed; experiments in which apparent foci of various special movements were found about the base of the encephalon, in the pons, medulla oblongata and other parts. The considerable amount of lesion often discovered at the floor of the fourth ventricle in general paralysis, and the degeneration of cerebral and spinal nerves, warn us against too ready an indictment of motor centres in the

cerebral cortex as answerable for the most frequent and characteristic motor impairment; that of the lips, tongue, face, and articulatory organs generally. But even in the production of these, the cortical lesion, if not the primary cause, is at the very least an important factor, nor can I agree with those who attribute this motor disorder and impairment to bulbar lesions exclusively. Here, too, the functioning of the centres is disordered, and voluntary movements that require accurate and complex co-ordination can no longer be deftly or properly performed. For not only is *transmission* of impulses to movement embarrassed, obstructed, and here and there absolutely blocked, by the products of hyperæmia, nuclear overgrowth, or inflammatory effusion, but by the same means, also, is the energizing of the brain as an organ of volition made irregular, fitful, and disharmonious—characters which are immediately imprinted upon the voluntary movements, the general activity. The speech and writing often display this to perfection.

Intimately associated with the disorder in the motor centres—is that perversion of the muscular sense, which, as Maudsley states, plays so important a part in general paralysis.

Yet as the disease progresses, and as the cortical centres become more and more dilapidated, as their efferent fibres become more involved in the lesion, and especially as the centres of the peripheral nerves in the medulla oblongata and cord become affected, there succeed to the inco-ordination, first a paresis of movement, and then a general helplessness. Sometimes the diseased condition of the spinal cord and, particularly, of the medulla oblongata, appears to set in simultaneously with, or occasionally even to precede, that of the cerebrum.

Rigidity and contractions of the limbs in the early stages of general paralysis, and especially those associated with convulsions or palsies, may be assigned to lesions affecting the cerebral cortex or the cerebral medulla;—in the later stages they may be due to the same, or to similar spinal lesions. Nevertheless, in explanation of the sometimes palsied, rigidly contracted, and spasmodic state of the limbs in the latest stages one may occasionally invoke the aid of the secondary degenerations already mentioned, especially the descending fasciculated consecutive sclerosis, intimately related to lesions of the anterior two-thirds of the internal capsule or its irradiations, and described by Türck, Vulpian, Charcot, Flechsig, and Pierret, and which is said to follow lesions pertaining to several groups, one of which consists of cortical lesions of some extent and depth situate in the ascending frontal

or ascending parietal gyri and contiguous parts, or in the paracentral lobule. But, we repeat, it is only rarely that one can assign any spasms, or rigid contractions, of the limbs in the latest stages, to these secondary descending degenerations in a nascent form.

Dr. Aug. Waller, in 1851, first described the wasting of the *nerves* consecutive to various injuries and lesions; and similar changes play their part in general paralysis.

Then, again, spinal myelitis or sclerosis may follow chronic spinal meningitis, a condition sometimes observed in general paralysis.

In the next place, to the inhibition or destruction, on the one hand, or, on the other, to the abnormal excitation of cortical motor centres, may often be assigned groups of symptoms which are extremely frequent in some cases of general paralysis. I refer to the localized, partial, incomplete, transitory, and variable paralyses so often seen, whether or not preceded by local convulsion or spasm. Of these, that leading to conjugated deviation of the head and eyes is one of extreme interest and unusual severity. I refer also to the local twitchings, the more or less localized spasms and convulsions, the general convulsions, the general muscular agitation, and the grinding of the teeth. These latter can be very closely reproduced by electrical excitation of portions of the brain-cortex in the lower animals, and, at least in one instance, in the human, and it is of great interest to trace the analogies between the effects of experiments of the physiological laboratory, and of experiments wrought by disease upon the same nervous foci. Most of the above are transitory, although, perhaps, recurring from time to time; others, however, are more frequent, or more persistent.

C. *Sensory Centres*.—Should one of the *sensory* centres, suppose the visual, be affected, there is primarily the rousing of ocular spectra, and even of hallucinations or illusional forms in accordance with the predominant nature of the thoughts and feelings at the time. The intellectual centres also react upon the sensory, and the excitement of cerebral circulation being general one would expect sensory disorder, even without *special* involvement of the cortical centres subservient to sensation. From the intimate association of sensation and thought in the genesis and ædification of mind, the only matter of surprise is that sensory disorders are not more frequent and marked in general paralysis. The partial distribution of the more extreme morbid changes is the plausible explanation of the fact.

Finally, like all the animal functions, that of the sensory

ganglia fails as the brain sinks in progressive degeneration, and not only the brain itself, but also the peripheral nerves and nerve expansions subservient to sensory functions, the secondary degeneration of which structures is usual in general paralysis, and is often manifest in the optic and olfactory nerves.

We may now for a brief space follow the fortunes, in general paralysis, of single, special, so-called cortical centres.

In examining the brains of general paralytics with reference to the doctrine of localization, it is necessary in the first place to ascertain the portions of nervous tissue more particularly diseased in each case. In this inquiry aid is usually derived from the special individual distribution of the *adhesions* of the pia-arachnoid to the cerebral surface. Habitually forming verbal or diagrammatic charts of these, we soon learn to recognize anything unusual or peculiar in their extent or distribution, in a given instance. In several cases, microscopically examined by the present writer with reference to this point, the portions of cortex underlying the special adhesions were more diseased than the cortex of neighbouring gyral summits, which, unlike them, was free from meningeal adhesions. In the investigation of this phase of the question of localization I have already, and elsewhere,* insisted upon the value of the precise distribution of these local adhesions of the pia-mater to the cerebral cortex, and of the changes in the latter associated therewith.

(a.) Of the cortical centres, the psycho-motor offer the most frequent and convenient opportunities of investigation with relation to this point; and of the motor symptoms, the convulsions and spasms, when present, have the greatest prominence and most dramatic character. To these will attention be confined in the first place.

Practically, however, it is only in the case of the more localized convulsive twitching and spasm that one can predicate the probable site of the lesion which brings about excitation of motor elements. When spasm spreads, when convulsion becomes more generalized, it matters not in what part it begins, or where alone it may for a time be manifest, or whether the parts first involved in convulsion are those which in health have been most in voluntary use,—the lesion producing that state of nerve-element which eventuates in abnormal excessive discharge, and is translated outwardly in convulsion—this lesion, I say, may in such a case occupy any one of a number of localities, or may be widely diffused. In fact it may not be in the cortex at all. Lesions in the medullary tracts of

* *Journal of Mental Science*, Jan. 1876, p. 567.

the brain, and in the downward continuations of the encephalon, may give rise to convulsion—at first localized, then more general. I have paid much attention to the above point, in general paralysis, and with the result of confirming the view I expressed some years ago (*loc. cit.*).

Nevertheless, many symptoms of the groups now under consideration can with more confidence be referred to circumscribed local excitation of the cerebral cortex, and are valuable for purposes of localization of brain-function. Such are, many at least of, the local paralyses and rigidities; such, also, are the local twitchings and spasms without loss of consciousness, now ceasing, now possibly transferred to another part, and anon returning to their primary site. Convulsion here is circumscribed, and is reduced to local convulsive twitching, or spasmodic distortion of several groups of muscles, or of one group, or of a single muscle,—convulsive twitching which may continue for hours or days without ceasing; may alternate with equally localized twitching or spasm elsewhere, and which constitutes one of the most striking features in general paralysis, but is only accessory and not invariably present.

These partial convulsions, as Louis Landouzy points out, may be produced by many diseased conditions affecting the frontoparietal region, such as tubercular meningitis, softening, hæmorrhage, “foyer” of capillary apoplexy, or of encephalitis, abscess, tumour, and traumatic injury. In this aspect the *nature* of the lesions is secondary, their *seat* everything. The more extensive of these convulsions for the most part accord with the description by Bravais, and later, by Hughlings Jackson, of three kinds of hemiplegic epilepsy: the convulsion beginning in one at the face, in another at the hand, and in a third at the foot, consciousness being usually retained, the voluntary power over the part affected often being not wholly annulled during the “fit”; and paralysis, confined to the same limits as the convulsive movement, ordinarily following the latter as the shadow follows the body.

The same symptomatic dissociation characterizes the cortical *paralyses* often seen in the affections mentioned above, and, like the local paralyses more commonly observed in “general paralysis of the insane,” they have the following characters. They are, at least at first, I., partial; II., incomplete; III., transitory; and IV., variable. Any of these characters may be absent in a given case. And, indeed, Cruveilhier long ago observed that circumscribed cerebral softening sometimes produced transitory local paralysis, or momentary loss of speech, or temporary stiffness, in this or that part of the body.

Again, contractions of the limbs frequently supervene in ordinary meningo-encephalitic affections, of a limited local nature and such as are followed by partial and progressive palsy. A similar continual cortical irritation occurs in general paralysis, and accounts for the more transitory recurring contractures sometimes seen therein, or even for some of the more permanent contractions of the limbs.

In the above groups of partial convulsions, spasms, and paralyzes, it would appear as if the experiments of the physiologist were reproduced by disease, and one looks for some definite local, and probably cortical, lesion in explanation. Vain search, too often, in general paralysis! Many cortical centres, as a rule, are markedly involved in obvious change, and when those extremely diseased are comparatively few in number they often are not homologous with those by irritation of which similar results are produced in the lower animals.

By augmenting the duration and intensity of an experimental excitation of a cortical centre in the motor zone, the convulsive movement, at first limited, may become general; and the same thing may occur in general paralysis.

And truly it must be confessed that the centres actually discharged, as represented outwardly in the convulsion or spasm of general paralyzes, are not necessarily those most obviously diseased; for, without effecting important structural changes in them, the intra-cranial morbid process may utterly disorder their functions by the disturbance it necessarily causes in their circulation and nutrition.

Again, as previously stated, the convulsions are not always of cortical origin: and it may be added that modifications in the constitution of the blood itself may here suffice to rouse unstable centres to convulsive action.

I might here relate at length the exact course, march, and distribution of many convulsive, spasmodic, and paralytic seizures in general paralysis, and compare them with the post-mortem records. But the recital would be tedious. The enormous difficulty has been that in the vast majority of cases the cerebral lesions are very extensive. In some of the cases wherein the lesions apparently producing convulsions and spasms were more localized I have been unable to trace a harmony between these and the results of physiological experiment; in other cases they have seemed to harmonize fairly.

I conclude * that convulsion and spasm, in general paralysis,

* As also in *Journal of Mental Science*, Jan. 1876, pp. 575, 576.

when of cortical origin, are sometimes due to localized cortical lesions, to which the distribution of the cerebro-meningeal *adhesions* often affords the clue, but that sometimes there is diffused cortical "irritation," which expresses itself outwardly in convulsions which follow one or other of several favourite courses, and begin in parts which are highly differentiated and whose movements are of the more special and voluntary kind; and that similar convulsions may sometimes be produced by "irritation" commencing in different parts.

To glance at other motor symptoms.

It is very easy, as some have done, to assign the characteristic motor signs of general paralysis, each to the lesion of a special part of a cortical motor zone, as mapped out by recent experiments, and to say that the dysphasia is due to lesion of this part, the ataxia of the limbs to lesion of that, and so on. But to speak thus is idle: to prove it is the labour. For years I have attempted to test this hypothesis; to formulate it is nothing. To formulate it and then rest satisfied, is but to join the lotus-eaters.

Whether, as Tamburini believes, and as Ach. Foville suggests, the pupillary modifications in general paralysis are due to excitation, to exhaustion, and to final destruction, of some cortical centre, or not, is, in the present state of science, a most difficult inquiry. Without being able to prove the affirmative there is ample reason to assume that it is true in some cases, and Viel has emitted a clinical and experimental study on the pupillary changes caused by artificially produced, local, meningo-encephalitis.

Of the local motor symptoms of cortical origin, occasional in general paralysis, one of the most interesting is the rotation of head and conjugated deviation of eyes—a symptom in cerebral affections to which Vulpian first directed attention. Probably, in some cases this is paralytic in nature—in others spasmodic;—being due, in the one case to the unopposed action of the healthy rotator centre in one hemisphere, that of the opposite hemisphere being temporarily put out of use by some lesion or inhibition; and, in the other case, to morbid unilateral excitation of the rotator centre.

Though possibly due to irritation of certain cortical centres, yet the persistent bending forwards of the head and neck, sometimes seen, is perhaps due rather to changes in the upper part of the spinal cord, and therefore, like the rigidity and contraction of the limbs in the latest stages, may be the result of secondary spinal and neural changes.

(β.) *Sensory*.—If the hippocampus major be proved to be

the centre for tactile sensation it would be of importance to note in relation to this, and to the failure of tactile sensibility, and the frequent convulsions, in the later stages of general paralysis, that Delays long ago found this part markedly indurated and atrophied in many cases of general paralysis, and that its induration and degeneration in chronic epilepsy have been described by Bouchet, Meynert, Hemkes, and others.

To lesion of the angular gyrus Crichton Browne would attribute the impairment of sight usual in the later periods of most cases; and to lesion of both angular gyri the complete blindness rarely observed. I have seen a crucial case in direct opposition with this hypothesis as to the blindness.

In general paralysis Voisin ascribes temporary blindness to encephalic congestion; permanent blindness to secondary neuro-retinitis, or compression of the nervous elements. And loss of smell to alterations in, and atrophy of, the olfactory nerves.

In conclusion.—Whether, in general paralysis, the principal mental symptoms can be entirely referred to the organic changes in certain frontal (and parietal) convolutions—the motor to those of the so-called cortical motor zone—the sensory to those of certain portions of the temporo-sphenoidal and parietal,—must remain a matter of question. And all the more so as, with some points of general accord, the conclusions of Fritzsche and Hitzig, Ferrier, Carville and Duret, Charcot and Pitres, Schiff, Albertoni, Michieli, Tamburini, Fürstner, Lussana and Lemoigne, show considerable, and in some cases irreconcilable, divergencies the one from the other.

That there is a localization of cerebral function is indubitable, but the rigid delimitation attempted by some recent investigators does not appear to be in harmony with the facts of nature. The action of one part of the cortex can be supplemented by that of another far more than some of them are willing to allow; there is more alliance than they admit between different cortical loci or centres which can operate towards the same result,—more of a capacity for the loose, flexible, yet effective, association of units, as of an army of men—not a rabble,—an association for the accomplishment of a given purpose. This or that one may fall out of the ranks, but the march of the host is not arrested nor its purpose stayed.

The mass of facts arranged by the masterly skill of Brown-Séquard, and found in opposition with certain recent doctrines of rigid localization, cannot lightly be either ignored or explained away.

Moreover, I think it cannot be without meaning that the

mental symptoms usually differ so much between themselves when the morbid process is earlier, and more severe, extensive, and persistent in one or the other cerebral hemisphere, and it may be inferred that the functions of the right hemisphere differ considerably from those of the left, although they are similar to so very great an extent. This, at least, is the result of an analysis of my own cases, a result not anticipated, and which came somewhat in the nature of a surprise. Yet to have been prepared for a revelation of the kind one need only have recalled to mind the observations of Brown-Séquard, Charcot, Callender, and Hughlings Jackson as to the different pathological relationships of the two cerebral hemispheres.

Besides the group of facts relating to aphasia and the more usual *habitats* of the lesions producing it, there are those upon which Brown-Séquard has based his view that of the two cerebral hemispheres the lesions of the right are (*ceteris paribus*) more frequent and fatal—give rise to more marked hemiplegia—more frequently are complicated with nutritive disorders (acute bedsores, sloughing, etc.) in the palsied parts—more often are evidenced by convulsion and tonic spasms of the limbs and by conjugated deviation of the eyes, and more often also by *direct* paralysis or convulsion (*i.e.* of same side as lesion). Hysterical paralysis, also, he, like others, found more frequent in the left limbs, and therefore generally with affections of the right brain. In cauterizing special parts of the cortex of certain of the lower animals he found the narrowing of the palpebral fissure, caused thereby, to be constant only when the right was the hemisphere operated on, and the results were quite different according to the side cauterized.

In regard to the relative fatality of lesions of the right hemisphere, and their more frequent association with convulsions and spasms, the view perhaps originated with Mr. G. W. Callender. Excluding those of the ventricles, basal ganglia, and peripheral brain-surface, Mr. Callender found hæmorrhage and other sudden lesions of the right cerebral substance to be followed by convulsion in a much higher ratio than similar lesions of corresponding ubiquity in the left hemisphere. In lesions of the parts specified he found sinistral paralysis almost always accompanied by convulsion or rigidity; dextral paralysis almost never so accompanied; and, concluding that the district the lesions of which more particularly gave rise to convulsion was that supplied by the middle cerebral arteries, he added that some part of the *right* cerebral hemisphere was specially connected with the occurrence of convulsions. Among the cases collected by him, of 48 cases of

palsy of the right side, only 7 also presented convulsion or rigidity; while of 61 cases of left-side palsy 39 presented either convulsion or rigidity. But excluding the cases in which the optostriate bodies were the parts affected the difference was still more striking, for then of 37 cases of lesion of the left hemisphere 7 were associated with convulsion or rigidity; and of 47 cases of lesion of the right hemisphere as many as 39 were accompanied by either of these symptoms. Callender also found the *average* duration of life after sanguineous apoplectic effusion in the right hemisphere to be only about one-eighth of the *average* duration of life after similar effusions into the left hemisphere. The above convulsions with brain-lesions belonged to one or other of three groups, one of which consisted of those just described; another, of those from extravasation, aneurism, tubercle, etc., along the track of the great vessels, especially the middle cerebral arteries; and another, of those which occur at the moment of hæmorrhage.

Long ago Dr. Sieveking, when describing epileptic seizures, said "it has appeared to me that the left side is the one most frequently affected." Thereby almost certainly implying a point of departure in the right side of the brain, or medulla oblongata above the decussation.

On the other hand, L. Landouzy, in the cases of local cortical lesions collected by him, found that about two-thirds were of the left hemisphere.

Hughlings Jackson suggested that the right hemisphere was the one leading in Perception; the left the one leading in Expression. Moreover, that the left posterior parts of the cerebrum and the right anterior parts were the substrata of *subject* consciousness; and the right posterior and left anterior parts were the substrata of *object* consciousness. Further, that lesions of the right posterior lobe impair the intelligence more than similar lesions of the left (causing imperception, *i.e.* defect of recognition). But this last is connected with his view that the posterior lobes of the cerebrum are the seat of the most intellectual processes, a view which is in direct opposition with the results of post-mortem examination of the brains of the insane.

I have found that when one cerebral hemisphere principally is attacked in general paralysis, the difference in relative damage to the two hemispheres being great, then the clinical phenomena almost always vary extremely with the particular hemisphere more affected. When it is the *right* hemisphere, exalted delusion, gaiety, expansive delirium, and maniacal excitement, predominate; when it is the *left*, either emotional depression, melancholic ideas and, perhaps, hallucinations:—or else an

extreme and early dementia, are unusually obvious, if not predominant. Thus the character of ideation, emotion, organic sensation, special sensation, of motor activity, and hence of general conduct are considerably different in the two cases, as a rule. Exceptions exist, possibly because in them it may be that the morbid process is long drawn out in one hemisphere; and is active, and the cause of striking disorder, though less severe and of shorter duration in the other, thus masking the effects of the former and graver lesion, until, or partly until, the period arrives when the progressive lesion entails a profound dementia, and ataxic and paretic helplessness, in which all, or most, of the other mental and motor symptoms are extinguished; or, again, perhaps because the parts to lesions of which characteristic symptoms are specially due are less involved than usual. These however are merely suggestions, in explanation of the above-mentioned exceptions, on which no stress is laid. Other exceptions are capable of explanation by the predominance of some one or more of those other conditions that rule the mental phenomena of the disease, and are discussed in a preceding section.

A word as to the cerebellum.

Though not markedly diseased, as a rule, in general paralysis, the cerebellum is often involved; its meninges become hyperæmic, thickened, and opaque; and near the median line it is sometimes the site of slight adhesion and of cortical degeneration. This has recently been denied; nevertheless it is the case. How far this diseased condition, when present, may give origin to the vague and purposeless movements, contribute to the general failure of power, and conduce to the imperfect maintenance of equilibrium, seen in general paralysis, or, possibly, even sometimes produce its convulsions, or its occasional headache and failure of vision,—it would not be easy to decide. Cerebellar changes, however, are not needed to explain the symptoms, and are probably of little importance here.

Nor is it useless to reiterate that some of the clinical phenomena in the course of general paralysis depend, or partly depend upon disorder or impairment of the medullary substance of the brain, the basal ganglia, the pons and medulla oblongata, the spinal cord and nerves, and last, though by no means least, of the sympathetic ganglia, especially of those in the cervical region.

Yet is general paralysis primarily and principally a disease of the cerebral cortex.

CHAPTER IX.

PROGNOSIS AND THERAPEUTICS.

PROGNOSIS.—The prognosis is necessarily of the very gravest. This was partially recognized even before “general paralysis” was differentiated in nosology, and when it was lost in the throng of “paralytic” affections. Thus so far back as 1820 M. Georget wrote, “la folie compliquée de paralysie ne guerit jamais.” Practically speaking, to detect the existence of decided general paralysis is to assign the patient to a comparatively early death. As soon as he is fully satisfied of the existence of true general paralysis it is the duty of the physician to say at once that the case is without hope, and *curative* art without reliable and permanent efficacy therein. Cures or recoveries of general paralysis have been reported it is true; but in view of the obscurities surrounding the diagnosis in certain of its forms the question has very justly and very often been raised whether the cases thus said, and doubtless in good faith, to be cured or recovered were or were not genuine examples of the disease. In some cases the so-called cures or recoveries have, in reality, been only temporary remissions of the relentless malady, which before long has attained the natural consummation of its course in death. To justify the gravest doubt and most cautious circumspection of reported cases of recovery from general paralysis, one need only bear in mind how considerable is the number, among the cases published as general paralysis during the past fifty-seven years, that have not been genuine examples of that affection; and one need only examine the mortality tables of many an asylum in which, through faulty diagnosis, general paralysis evidently stands accused of ravages not its own. And yet it is not at all rare to find instances of highly pronounced remission, or, perhaps, of apparent complete disappearance of all mental symptoms; and sometimes, on a superficial examination, of the disorders and defects of motility as well. I say, on a superficial examination—for almost always, if not always, after active exertion, and upon a protracted and accurate investigation being made, the speech, or the tongue, or the lips, will betray that fatal ataxia, which to the practised ear and eye cries “no recovery”.* Though no longer

* Shakespeare, *Troilus and Cressida*, act ii. scene 3. Indicated by Dr. T. Clay Shaw.

"plaguey proud,"* but on the contrary mentally recovered, the patient still bears on him more gravely ominous "death tokens"† in those delicate lesions of motility.

Two only of Bayle's six cases of "amelioration or recovery" in general paralysis can for a moment be accepted as recoveries, nor even then without hesitation. Delaye quoted a case from Esquirol's clinical course in which cure followed when spontaneous hæmorrhoids became established. On the authority of Calmeil we have it that, after twenty years' experience at Charenton, Royer-Collard had never seen a case thoroughly recover, and that Esquirol only spoke of three exceptional instances of cure. He himself added two, of which the second at least was evidently a case of well-marked remission only; and in 1859 he spoke of the extreme rarity of true cures of general paralysis, notwithstanding that the disease may remit, and remain stationary during ten or fifteen months, or nearly two years.

Lélut, Ferrus, Falret, Trélat, Guislain, Bonnefous, Fabre, Foville, Laffitte, Combes, Lunier (6), Pinel (*neveu*) (2), Bulard and others believed they saw cases of recovery or cure. Baillarger mentioned a number (9) of recoveries, including some of those just cited, and also cases by Earle, Renaudin, Beaume and Morel. It has, however, been suggested that Baillarger's so-called cases of cure were in truth only examples of *mania plethorica*. Billod described in all detail the case of a man aged thirty-three years whom he thought to be cured. Five, at least temporary, recoveries were alleged by Brierre de Boismont. Two general paralytics mentioned by Dr. R. Boyd were discharged recovered, "but after several months both relapsed." Another had "recovered from the paralysis (!), but was still a patient in the asylum." Several cases were tabulated by Burman as having been discharged, on recovery, from an English County Asylum; some of whom returned to die; the further history of others was unknown; and one had not been insane (!). Even assuming the diagnosis to be correct in all, there is no proof that these were more than instances of remission of the disease. Dr. S. W. D. Williams mentions a case of recovery, and this was confirmed by examination nearly three years after the discharge. The patient, a male, aged twenty-five, was discharged recovered, twenty months after the appearance of the first symptoms. Mercury had a place in the treatment employed.

* Shakespeare, *Troilus and Cressida*, act ii. scene 3. Indicated by Dr. T. Claye Shaw.

† Ibid.

Dr. Hack Tuke refers to two cases of apparent recovery under the care of Dr. Macleod.

Cures have recently been reported in Germany by Moriz Gausster (2), by Schüle, and by Flemming.

I find that several cases, in addition to some of those I had collected and have mentioned above, have recently been cited by Doutrebente. These are cases, of supposed recovery, by Bouilaud, Marcé, Delasiauve, Dubuisson, Thos. Willis, Védie, S. Roy, and L. Meyer (Meyer's were eight cases, one relapsing).

Dr. Wm. Macleod, writing immediately after the patient's discharge, records a case of mental recovery enduring at least eight months, but says there was still "a certain slowness of speech, and when excited in speaking about himself a slight thickness of speech is observed:"—this was only a remission, therefore.

Among my own patients one was discharged completely recovered mentally, in whom only the faintest occasional traces of some of the physical signs persisted. He remained well for some time, but finally mental symptoms again came on.

Another, discharged, mentally recovered, and bearing only the slightest and very doubtful trace of the motor order of symptoms, continued thus for ten months or more. This was the second attack, and had followed a highly pronounced remission or recovery of shorter duration than the above-mentioned second one. He has since relapsed for the second time, and therefore into his third attack of well-marked general paralysis.

Of a third patient, discharged in a similar condition in Nov. 1877, I have not yet heard any tidings. All traces have been lost of a fourth and of a fifth who were discharged, with less absolutely complete indications of temporary recovery than existed in the preceding patients.

It is not clear upon what is based the statement of Doutrebente that the prognosis is always more grave when the disease is caused by alcoholic or venereal excesses.

It is noteworthy that several of the above-mentioned instances of recovery or of prolonged remission have supervened on accidents, violent injuries, or diseases of such a kind as to produce lively so-called revulsive effects. In exemplification of this one may refer to cases of apparent recovery following upon erysipelas, or profuse suppuration, or burns, or lumbar abscess, or amputation of the thigh, or fractures of the tibia, and phlegmonous inflammation of the thigh.

A wild and troublesome general paralytic recently under my own care was much better in mind when, and for a time after, he

was laid up with a compound fracture of the tibia, which he incurred by jumping from a wall in an effort to escape.

Guided by these indications, I have often thought it might be beneficial to create large ulcers or suppurating surfaces, especially on the lower extremities of general paralytics, and to maintain a prolonged and free discharge therefrom, but I have not yet had the courage to put the plan into actual operation. And this leads to the consideration of therapeutics and hygiene as adapted to general paralysis.

THERAPEUTICS AND HYGIENE.

Prophylactic. (I).—The true prevention of general paralysis would consist in :—

(*a.*) A regulation of marriage by the pressure of enlightened public opinion, so that it should be esteemed a grave social offence to intermarry with those whose hereditary tendencies are undeniably neuropathic, and whose children would be predisposed to general paralysis.

(*β.*) And in good education, both moral and intellectual, so as to render the various psychical powers strong, well balanced, and harmonious in action; adapted to resist hostile influences; and to promote that self-restraint and self-contained reaction upon the social, religious, and intellectual environment, that conserve the mental energy, and fitly direct the dynamic powers of the whole being. It goes without saying that the above implies an avoidance of all excesses, of over-strained and extreme emotion, or of excessive intellectual labour. It is easy to prescribe all this; it is more than difficult to live up to it, and to induce others to do so.

II. Of more practical use is it to devise a prophylaxis and therapeusis adapted to arrest the course of an already threatened general paralysis.

A patient threatened with general paralysis, one who has shown symptoms of the prodromic period,—the period of mental alteration not yet amounting to decided mental alienation,—should be at once freed, as far as possible, from the conditions under which symptoms of so sadly prophetic a nature have arisen. A perfectly regular life, early hours, moderate and regular bodily exercise, a total disuse of alcohol in any form, and of tobacco; the use of bathing and friction of the skin, the application of cold to the head when it becomes unduly heated, while the feet are kept warm and if necessary mustard, or hot, pediluvia are

used, together with the maintenance of a perfectly soluble state of the bowels,—all these should be enjoined.

Every source of mental worry, anxiety, annoyance, fear, chagrin, should be scrupulously avoided, at almost any cost. All intellectual labour should cease, and just such an amount of reading, of conversation, and of thought, should be undertaken as will afford the most gentle of intellectual and emotional exercise. The society should be that of the patient's family or intimates. In a word, the patient must go out of his ordinary life, must retire from his duties, labours, contrarities, turmoils, and ambitions, and in repose seek a renewal of nervous tone, and of power of resistance to hostile influences.

III. Treatment of confirmed general paralysis.

For the most part the means suggested in the treatment of the prodromic period will still conduce to amelioration. In so malignantly fatal an affection, general management and nursing hold the chief place in the treatment.

Most general paralytics should be removed from home for a time, either to an asylum, or to some house of which part can be set aside for their use. To retain them at their own homes, where they may squander their means, are most dictatorial, fly into furious passion if not obeyed, and where everything rouses their desire to alter, sell, or destroy, is to promote angry and disturbing scenes, most prejudicial to the patients, and most painful to those about them.

Tact and gentleness are very necessary in their management:—by tact, gentleness, and *bonhomie* many of them may be kept in good-humour; or, at least, those angry outbursts which, too often, follow the slightest crossing or thwarting, may be avoided, as far as possible. In the early stages, peace and mental rest should be sought by every means, but too often are unattainable, and when furious paroxysms of destructiveness or violence occur a judicious isolation or seclusion may occasionally prove beneficial. In the early stages, also, and especially in the forms characterized clinically by much mental excitement, by expansive delirium, or by so-called congestive tendencies, the nourishment should be light, easily digestible, limited, and absolutely free from alcohol. This limitation of diet is far from being always easy to carry into effect, inasmuch as the patient's appetite is usually large, nay, often voracious, although when this is more apparent than real—the patient eating over-plentifully through inattention—it may be prevented by judicious management. In such cases, also, the excretory organs, and especially the bowels, should be kept in free action by the usual dietetic and other means. As far as possible

the patient should live in the open air and take a moderate amount of exercise, while daily warm baths to the body, simultaneously with cold applied to the head, are often of service.

With the advancing progress of the disease the diet may be made more generous, and in the third and fourth periods may with advantage be extremely liberal. Alcohol, which as a rule does so much harm in the first and second periods, will sometimes aid in prolonging life when administered in the fourth and in the latter part of the third period.

When the patient becomes unable to walk by himself with safety he must have the assistance of attendants in taking regular daily open-air exercise; at a later period he may recline in an easy-chair, sitting on air-cushions; and thence, either by a gradual decline, or after convulsive or paralytic attacks, he must sink yet a step lower in the scale, and pass his days on a water-bed. But usually for a short time only. Bedsores tend to form, pulmonary affections and diarrhœa afflict his feeble and now attenuated frame, the excrements all pass involuntarily beneath him, and, as a rule, the end is not far. Hence, in the later stages, the paramount importance, next to good feeding, of perfect cleanliness and of the prevention of bedsores. These are only attainable by the most constant and scrupulous care in the instant removal of all excreta, the use of "railway" urinals, where possible, and of a water-bed, frequent changes of position, and the employment of a hardening application on the parts exposed to pressure. After a comparative trial of a number of such applications I prefer a strong saturnine lotion.* Sometimes, however, the bedsores are *acute*, (*decubitus acutus*), cerebral or spinal in origin, and absolutely unpreventible. More special details of the treatment any of these require are given below.

Throughout, the solubility of the bowels should be maintained. If they are inert, strychnia may be used as recommended by Verga, but in the later stages diarrhœa is often troublesome and obstinate, and, like other incidents, accidents and complications, —such as lobular pneumonia, nephritis, hectic from bedsores, and pyæmia,—must be treated on general principles.

By these means, and by those detailed below, a patient will sometimes be enabled to survive for more than a year after the commencement of a continuously bedridden state. Of course, I refer to a bedridden state entirely due to the disease itself and not to any accident or complication.

* Plumbi Acet. 3i; aquæ, oi: or Liq. Plumbi Subacet. (B.P.) sufficiently diluted.

Little need be said as to the *medicinal* treatment of general paralysis. The general and local treatment of the disease itself, will first be referred to; and then that appropriate to certain complications.

Pharmaceutical, and other, Treatment of General Paralysis.

—The earlier writers on this subject enjoined the employment of an active antiphlogistic therapeutics. Those were especially the days of its treatment by low diet, bleeding, leeching, cupping, purgatives, moxas, vesicatories or setons to the nape or elsewhere; mercurial inunctions, antimony, diuretics, and by cold to head with warm bath to general frame. Possibly, in this country, treatment of this kind has suffered undue neglect of late. Bleeding, for example, might prove useful in some cases in which very obvious cerebral hyperæmia is present in the early stages, or where violent congestive seizures occur. Yet venesection could only be employed exceptionally here, and I have no personal experience of its use.

Counter-irritation or revulsion by blisters, suppurants, cauteries or setons to the nape, the spine, or the scalp, have also perhaps fallen into too great desuetude in this country, and are sometimes of service in the middle and later stages of the disease. Some foreign physicians still make active use of these means, and of venesection, and of leeching the lower extremities.

Another old method may be revived with advantage in certain cases; that by which leeches were applied to the nose, anus, or vulva, when habitual sanguineous losses had been checked at, or before, the incidence of general paralysis, according as the losses had been those of epistaxis, of hæmorrhoids, or of the menses.

Nothing but evil may be anticipated, however, from one old plan of treatment;—that by which Royer-Collard and Bleynie administered large doses of tartarized antimony.

We need only refer to the systematic employment of venesection and leeching, especially in the earlier periods; or of repeated blisters to the head and spine in acute, galloping cases; or of repeated cauterization of scalp, nape and spine, or nuchal seton, in chronic cases, and particularly when the spinal symptoms are marked. Or, to the protracted use of digitalis or digitaline, of the haloid bromides, of quinine, of physostigma, of purgatives, of extremely prolonged baths, or of short daily, or even bi-quotidian, cold baths.

Mercury and potassium-iodide have also been recommended in the treatment of general paralysis. They often relieve any early pain in the head or extremities, but I have rarely seen life decidedly lengthened by their use, or the course of the disease, even

temporarily, arrested. I refer to pure uncomplicated cases of general paralysis. Should the patient, however, be also syphilitic, or should the differential diagnosis between cerebral syphilis and general paralysis be uncertain in a given instance, it is well to treat the patient for a short time with mercurials and for a longer with the iodides; carefully watching for, and energetically following up, any resulting improvement that, happily, may shed its ray of light athwart our gloomy prognosis or our dark perplexity.

In the early stages, if the patient is acutely maniacal, or if paroxysmal outbursts occur, benefit may be derived from the use of quickly-acting purgatives, and of warm baths of from fifteen to thirty minutes' duration, with cold applied to the head simultaneously by ice-cap or cold-water cloths or cold-water douche. In these conditions potassium or ammonium-bromide, veratrum viride, and physostigma (C. Browne) sometimes prove of use; and æther-spray to the head may have a calmative effect, as I have found. In other cases Tr. of digitalis (℞ xx-xxx every four to six hours), or digitaline, as recommended by Voisin, acts well; but I have found that opiates, though often extolled, do mischief in this phase of the disease. Austin advocated large doses of extract of hyoscyamus here (grs. xx-xxx, ter in die). I cannot speak strongly in favour of the tincture in large doses: with potassium-bromide it occasionally suffices.

The preceding remedies are for the most part employed during the daytime. But during the high tide of excitement and sleeplessness in general paralysis a moderate or a full dose of chloral-hydrate, or of a mixture of chloral-hydrate and potassium-bromide, at night will often prove beneficially hypnotic. The latter may occasionally be given more frequently; I* have observed that it does not necessarily lower the *average* temperature under these special circumstances. Hypodermic injections of morphia and draughts of hyoscyamine have also been recommended for the insomnia and night restlessness, and the latter has sometimes proved efficient in my own practice. (Merck's amorphous hyoscyamine gr. $\frac{1}{4}$. See also R. Lawson.)

But in the absence of great excitement and sleeplessness or of the maniacal paroxysms, it is my usual practice to prescribe either veratrum viride or certain tonics, according to the condition of the patient:—the veratrum viride if, together with moderate excitement and evident cerebral hyperæmia, the general frame and visceral health are robust:—the tonics if the patient is calm and quiet, or demented, from the first, or becomes so during the

* *Practitioner*, June, 1874, p. 429.

course of the disease, and particularly if he is enfeebled, emaciated, exhausted, or phthisical; and then, indeed, whether he is agitated or not. Nevertheless, even in the former class of cases the time for tonics and restoratives is not long delayed, and during the whole of asylum life the system of most general paralytics responds favourably to their effects. Of those I have used the best results have been derived from liquor ferri perchloridi (℥ x-xx ter in die) taken immediately after each meal. If any tendency to constipation manifests itself twenty grains of magnesium-sulphate may be added to each dose; or relief may be obtained by enemata; or by the use of podophyllin occasionally, or of one-fifth of a grain of it every morning or more often, the podophyllin being employed either alone or with strychnia and belladonna; or, by the substitution of citrate of iron and ammonia for the perchloride.

In the calmative agents, already referred to, during phases of excitement, in veratrum viride during the earlier periods of some few cases, and in perchloride of iron, either throughout, or in the later stages only, have I thus far found the best pharmaceutical treatment of the disease itself *during those portions of its course which the patients usually pass in a lunatic asylum*. The complications also require treatment, and this is described below. To the chalybeate, cod-liver-oil or arsenic* may often be added with advantage, especially in the later stages. Taken collectively, the general paralytics under my own care have hitherto derived more advantage from a solution of the perchloride of iron† than from all the other forces of the pharmacopœia invoked on their behalf, and its effect in prolonging life is often very decisive.

IV.—*Treatment of the more Important Complications in General Paralysis.*

THE EPILEPTIFORM SEIZURES OF GENERAL PARALYSIS.—In the severe epileptiform seizures it is well to give chloral-hydrate, either alone or with potassic bromide, and usually by enema, but sometimes by the mouth. If by enema, the parts should be well plugged to prevent its escape. If the patient can swallow it is better to give the chloral by mouth. This plan I have used with epileptics from time to time since 1871.‡ If the *status epilep-*

* Arsenic I have sometimes used in general paralysis: recently it has been eulogized by Dr. Adriani.

† Liq. ferri perchloridi (B. Ph.).—Or, solid perchloride ʒii; water fʒxvi.

‡ See also J. A. M. Wallis on the use of chloral-hydrate in the convulsions of general paralysis.—West Riding Reports, vol. v. p. 257. To him seems due the credit of introducing this use of the drug in general paralysis.

ticus is established inhalations of chloroform may be resorted to, or the enemata just mentioned;—and subsequently the latter with brandy. In any of these cases I generally use thirty, and never exceed forty, grains of chloral-hydrate in a single enema, but watch closely, and repeat the enema as may be required. Cold may also be applied to the head by ice-bag or evaporating lotion; the bowels may often with advantage be first cleared out by a clyster of sodium-chloride or of magnesium-sulphate and turpentine, and the patient be afterwards supported by nutritive enemata of milk, beef-essence, and, if necessary, of brandy.

Nitrite of amyl has failed in my hands here.

THE APOPLECTIFORM SEIZURES OF GENERAL PARALYSIS.—In the apoplectiform attacks, elevation of the head; free purgation; cold applied by ice, or in various other ways, to the head with, or without, a prolonged warm bath to the rest of the body; and ammonium bromide or potassium bromide and ergot in full doses, will often prove to be favourable ordinances.

In these conditions, also, and whenever cerebral congestion is marked, it is often suggested to resort to venesection, to apply leeches freely to the nostrils, mastoid processes, anus, vulva, or to the lower limbs; and when the congestive tendency is obvious, but apoplectiform symptoms have not yet arisen, to use very hot or mustard pediluvia. Here, also, digitalis, quinine, and cold baths have been extolled.

THE BEDSORES OF GENERAL PARALYTICS.—In the management of the *bedsores*, prevent them as far as possible by perfect cleanliness, by placing the patient on a water-bed, by frequent changes of his position, and stratagems devised to prevent pressure upon threatened points, and by the application of strong lead-lotion. If, in spite of these precautions bedsores form, or if they are *acute*, and therefore not preventible, apply a solution of carbolic acid or of potassium-permanganate and over it a linseed poultice, cut away sloughs as soon as they are loosened, syringe out regularly, frequently, and thoroughly with the carbolic, permanganate, thymol, or chloralum lotions, then dress with the same or with a solution of boracic acid, or with powdered zinc oxide; stimulate the granulations if necessary; and let the breach attempt to close under zinc ointment, or boracic acid lint.

So long ago as 1858 Brown Séquard* proposed a plan for the prevention of that sloughing over the sacrum and nates which is so often found in cases of spinal disease or injury, and which is closely allied in its pathology with many an acute bed sore in

* "Phys. and Path. of the Nervous Centres," Am. Ed. 1860, p. 260.

general paralysis. Two poultices were alternately applied—the one of pounded ice—the other a very warm bread or linseed poultice. The pounded ice, contained in a bladder, was applied for eight or ten minutes; the warm poultice for an hour or two, or even a longer period.

In similar cases Brown-Séquard also suggested the use of powerful galvanic currents. Spencer Wells, in 1848, advocated a weak continuous electric current in the treatment of ulcers.

Dr. Warren of Dublin also describes the treatment by galvanism of the bedsores in protracted spinal cases. This, he says, was first brought before the profession by Dr. Crusel of St. Petersburg and had succeeded in the hands of Hammond, Spencer Wells, and others. "The method of applying it was simple; a clean silver plate of a size corresponding to the ulcer was placed over the sore, a zinc plate, under which was placed a piece of chamois-leather wet in vinegar, having been laid on some part of the neighbouring healthy skin. The current was completed by a copper-wire joining the zinc and silver plates."

These several procedures deserve a fair trial for the prevention or cure of the bedsores occurring in general paralysis.

Catheterization should be avoided if possible; yet the bladder should not be allowed to remain distended and unrelieved. Before using the catheter a stream of strong carbolic lotion should be poured through it and its exterior should be smeared with carbolized oil. When the urine is turbid, ammoniacal, and mucous, the bladder may be washed out with a solution of quinine; or with a lotion of very weak nitric acid; or with very weak carbolic acid or ferric perchloride injections; and at the same time boracic acid or sodium-biborate may be given internally.



PART II.

CHAPTER X.

THE VARIETIES OF GENERAL PARALYSIS OF THE INSANE.

THIS chapter consists of excerpts from a paper published by myself in the *Journal of Mental Science* for April, 1878, p. 25 ; with a few slight verbal modifications or interpolations.

In any large group of cases of general paralysis, there are so great differences in the mental symptoms, the paretic signs, the mode of onset, the course, intercurrent affections, duration, variability, and mobility of symptoms, and pathological anatomy, of the several cases, that one must feel that there are varieties of the disease. This has led to subdivisions of the affection, or grouping of its cases, scarcely any of which have had a pathological basis. True it is that Bayle, who was the first to separate and clearly describe general paralysis, and who attributed its striking phenomena each to a special morbid change, was led to place his cases in five series ; in the first of which were simply the lesions of chronic meningitis ; and in the second abundant serous effusion was added to these ; while, in the third, consecutive inflammation of the grey cortex, in the fourth, arachnoid false membranes (cysts), and in the fifth, various cerebral affections, complicated the chronic inflammation of the soft membranes. Yet many of his explanations are obviously incorrect, although his work marks a marvellous advance in science. Again, Baillarger, Requin, Prus, Duhamel, Duchenne, Sandras and others, held that general paralysis occurred either with or without insanity, and the first-named of these denied that the insanity is anything more than secondary and accessory in general paralysis of the insane. Further, he speaks, and in this is followed by Lunier, of the symptoms of general paralysis as being produced by (1) chronic meningo-encephalitis, and by (2) chronic hydrocephalus (Dance, Moulin), or serous effusion following apoplexy (Rochoux, Moulin), or symptomatic of organic lesions. But they do not in any way distinguish between the cases arising from the two kinds of lesion, further than that Lunier asserted an absence of trembling of the limbs in those from lesions of the second kind.*

But nearly all the attempts hitherto made, as far as I know, to delineate shades or varieties of general paralysis, have but set forth and emphasized certain symptoms, and especially certain mental features. It is evident that this cannot correspond with any real division of the disease into true pathological varieties, and can, at the best, but play the rôle of a measure of clinical and descriptive convenience. To illustrate the statement just made, it is only necessary to refer to examples of the numerous subdivisions that have been made of general paralysis into semeiological forms.† Thus it has been divided into the expansive, the depressed, and the

* In his Treatise, published a year later than the paper from which the above lines are taken, Volsin describes five forms of general paralysis :—(1) The acute form. (2) The common form, with expansive ambitious delirium. (3) Paralytic dementia. (4) Senile form, connected with arterial atheroma. (5) Spinal form.

† For examples see original paper, *Journal of Mental Science*, April, 1878, p. 26.

maniacal forms; or, into paralytic mania, paralytic melancholia, and paralytic dementia; and these have again been subdivided.

Nosological divisions, based upon mental symptoms or psychological differences, as in the systems of Arnold, Pinel, and Esquirol, are confessedly of but temporary use, and not in correspondence with true pathological variations. Applied to general paralysis, they are even more inexact. For in the latter affection, even more than in ordinary insanity, the mental symptoms often undergo most decisive and, perhaps, sudden changes. There is in it a great variability, so that he who suffers from ambitious delirium to-day, may to-morrow exhibit symptoms like those of general mania, may soon afterwards be plunged into the depths of hypochondriacal woe, and ere long have left all hope behind, having irrevocably entered the portals of a progressive dementia. True it is that many retain almost throughout some predominating general character of mental symptoms; there are those who throughout specially exhibit either grandiose delirium, or symptoms allied to general mania, or to dementia, to hypochondria, to melancholia, or to circular insanity. True it is, also, that in cases such as these, there is a general tendency to certain differences in the morbid anatomy of the several groups. At least the facts of the morbid histology observed for several years past seem to warrant me in making this assertion: the present, however, is not the place or time to speak of this aspect of the question. Yet, for the reasons above mentioned, it seems that one cannot establish any real varieties based upon the differences in the mental symptoms.

Whether we shall ever arrive at them or not, I think the only true classification, subdivision, and terminology in mental diseases must be based upon the morbid alterations of tissue or of function of tissue which engender the symptoms—that, in a word, they must be anatomico-pathological. For these purposes there can be no real finality, no pathological exactitude, in semeiology or in aetiology. Moreover, it is desirable to arrive at a knowledge of the real lesions in the various groups of cases of general paralysis, and thereby assist in the elucidation of other departments of the study of mental diseases. For the very complexity and gravity of its morbid lesions render general paralysis, in one sense, a vantage-ground for the further pursuit of investigation into other domains of mental alienation. Indeed, it was once said to be the only mental affection which enjoyed the sad distinction of possessing a morbid anatomy. But that was not recently.

With reference to the great diversities one finds in cases of general paralysis, it is desirable to inquire whether groups of cases cannot be formed corresponding with certain differences in, or modifications of, the pathological lesions proper to the disease, each group of which shall present its more or less characteristic *ensemble* of symptoms, or its individual method of association and relation of symptoms. Early in 1873 I began an investigation with this object in view; but the material did not accumulate very rapidly, for some general paralytics were discharged during remission or (temporary?) recovery at the request of friends, and the friends of some declined to permit post-mortem examination.

General paralysis varies much in different cases as to the parts of the encephalon especially attacked; the disease has in this case spent its force with greater severity upon certain localities, in that case upon certain other foci, and in still another upon a third locality or group of localities. In endeavouring to localize the points of principal morbid implication, I began, in 1873, to specially observe and record the exact localization of all the principal adhesions of the pia-mater to the cortex of the cerebral hemispheres. This adhesion I had always looked for, and recorded in outline, in earlier necropsies, and had come to view it as the most characteristic naked-eye appearance in general paralysis. These views as to its importance were confirmed by microscopical verification, in several cases, of a greater intensity of cortical disorganization corresponding with the areas of more decided adhesion of the pia-mater to the surface of the brain, with limited separation of irregular layers of the grey cortex adhering to the meninges when these are removed (adhesion and decortication). In the early part of 1875 I wrote a paper (*Journal of Mental Science*, Jan. 1876), giving details of one of the cases in which each adhesion was thus recorded, and an attempt was made in that communication to see how far

certain of the symptoms observed during life could be explained by a reference to the irregular distribution of the adhesion and decortication, and how far, in that case, the results of the experiments of disease tallied with those of the experiments of the physiological laboratory. Perhaps, and as far as I know, the above were the first investigations into the exact localization and distribution of the adhesions in various cases of general paralysis, for the purpose of throwing light upon certain clinical features of the disease. I believe that valuable results are to be obtained from this line of inquiry, and, in publishing necropsies, I will describe the localization of these adhesions with minuteness. Yet is it true, as remarked by Bayle, that the adhesions may occur early in the course of the disease, and may remain a long time inactive in the production of symptoms. That only the *summits* of the gyri are usually the seat of the adhesions, the elder Foville explained by their being the parts most exposed to the immediate effects of the compression resulting from inflammatory turgescence of the brain, in the early stages of general paralysis. Able papers on these adhesions have recently been published by Drs. Crichton Browne and Achille Foville (*file*).

But the adhesions in question, and the associated changes wrought in the corresponding portions of the grey cortex, are not invariably present in general paralysis, and they constitute but one of the points to be examined in investigating the anatomical distribution of the more extreme degrees of morbid change in individual cases of that disease. The other conditions of the grey cortex must also be particularly observed, the portions of it earliest or most specially attacked must be sought for, and its state, both general and local, as to vascularity, consistence, bulk, pathological products, and degeneration, examined. An investigation of the same kind must be directed to the series of central ganglionic masses near the base of the brain, to its medullary substance, to the bulbar tissues, to the cerebellum, and to the spinal cord and its meninges, as well as the sympathetic ganglia. So, also, must be taken into account such secondary products and complications as excessive serosity, arachnoid hæmorrhage and cysts, pia-matral hæmorrhage, local pachymeningitis, local ramollissement.

Finally, there is required a careful microscopical examination of the nervous systems of a large number of general paralytics, carefully compared with detailed records of the clinical features observed in each. My leisure has been insufficient for this last undertaking.

But I have observed such marked differences visible to the naked eye, or obvious upon making a less elaborate or extensive microscopical examination, that it seems to be desirable to place some of the cases on record, grouping them according to certain differences in the pathological lesions of the encephalon, and to indicate the clinical features which in their totality constitute, as it were, the garb each group severally wears. I have long thought that under the name general paralysis (of the insane) were included several pathological varieties, in a manner analogous to, but less decided than, that by which formerly the name "pulmonary phthisis" shielded several varieties of pulmonary disease; and by which, again, "Bright's disease" included a number of more or less distinct morbid states. I do not offer the following groups as representing proved varieties of general paralysis, or even as by any means covering the whole of the ground in their clinical aspects. But if we can show that in general paralysis cases can be placed in groups, the members of each of which have a considerable similarity to each other in their symptoms, course, duration and pathological anatomy, then has a step been taken in the direction of establishing pathological varieties of that disease. Some accident or intercurrent malady may cut off a general paralytic before his time, and the pathological lesions may appear different from what they would have been had the affection run its usual course. This is, of course, true of all organic maladies. Avoiding such fallacies as would arise, for example, from considering that in the same case ending untimely or ending maturely we had two varieties, it is possible, I believe, to indicate several groups which possess a certain individual distinctness. Five of these will now be referred to. It is not meant that the essential pathological process is distinct in each. The separation between some of them is mainly based upon the differences in

the encephalic localities affected in each (3 and 4). In another instance (group 2) the difference seems to be mainly one of chronicity and mildness; but also of locality. In another (5) the local cortical induration appears to indicate a local morbid process, differing somewhat from that which is at the basis of the more usual change of the same part in general paralysis.

The pathological and clinical features of the five groups are described in detail at pages 31 to 43 of the *Journal of Mental Science* for April, 1878. Of that description I will here offer only the following very brief and necessarily imperfect summary.

FIRST GROUP.—*Principal Changes.*

1. Cerebral hypersemia and softening are observed, are more than usually generalized, but particularly affect the cortical substance of the superior, external, and, to a less extent, internal fronto-parietal regions.
2. The cerebellum is usually affected in a considerable degree, so are the basal ganglia, while the mesocephale and spinal cord also are apt to suffer, but in a less degree.
3. Adhesion and decortication are usually well-marked, are mostly confined in the cerebrum to the upper and external surfaces, are particularly well seen over the frontal lobes, are well seen on the parietal, less over the temporo-sphenoidal, and sometimes slightly or moderately over the internal and inferior surfaces. The cerebellum is often affected with adhesion and decortication.
4. The above changes are nearly, or quite, symmetrically disposed in the two cerebral hemispheres.

Principal Clinical Features.

1. There is variability of the mental symptoms, both intellectual, emotional, and moral.
2. Exalted or extravagant delusions are the most marked feature, while maniacal excitement and insomnia are frequently observed.
3. Gaiety, self-satisfaction, benevolence or pride are evinced; or the patients are selfish, haughty, hostile, obstinate, abusive; or destructive, untidy; and the habits often are filthy.
4. Transitory depression, or melancholia, sometimes comes on.
5. Dementia is occasionally the predominant mental character from the first.
6. Motor ataxy and paresis are present, are sometimes well-marked; but in the earlier period are often masked by the maniacal state, or but imperfectly developed. Motor restlessness is frequent.
7. Occasionally, epileptiform or apoplectiform seizures, choreiform movements, or tremor coactus are observed.
8. Now and then are there hallucinations of hearing or of sight. Later, are found defects of general or special sensation, or hypochondriacal sensations.
9. The average duration is short, being 11 months;—or about 16 months, including the first period.

SECOND GROUP.—*Principal Changes.*

1. There is atrophy of the brain, and considerable increase of intra-cranial serum, the ventricles are dilated and much granulated. The gyri of the brain are wasted, especially on the upper surface and at the frontal region, the corresponding grey cortex being either softened, or, occasionally, of about normal consistence, pale, watery, sodden, or at times of fair colour, or even mottled by irregular hypersemia.
2. Adhesion and decortication, usually slight or moderate here as compared with

the other groups, are principally at (1) the Sylvian fissures, (2) upper frontal, and (3) parietal, surfaces, and (4) base (orbital or temporo-sphenoidal). This is the order of relative degree in which the several localities are affected with the adhesions.

3. The white cerebral substance, softened in some cases, more or less indurated in others, usually tends to pallor.

4. The basal ganglia are generally pale, soft, shrunken; the pons and medulla oblongata pale and soft; the spinal cord may be more or less softened or indurated.

5. The meningeal changes are very marked, and extend to the base, and, like the other changes, are symmetrical.

Principal Clinical Features.

1. The mental symptoms in the earlier periods may consist partly or slightly in exaggerated notions, or paroxysmal excitement with strange demeanour, or, though rarely, exclusive dementia predominates from the first. Subsequently, there is a protracted stage of dementia in which fitful outbursts of excitement, or hypochondria, may occur.

2. The quiet self-satisfaction, or the negative emotional state, of the early periods, is usually replaced by morose, peevish, distressed, or apprehensive states of feeling, and these by obliteration of the emotional life. The habits become foul; the patients often are destructive, obstinate, abusive, brutish.

3. Motor paresis is comparatively slight in the earlier stages, and of slow progress, gradually becoming more marked, especially in the lower limbs. The patients are usually bedridden a long time, and often grinding their teeth.

4. There is peculiar absence of epileptiform and apoplectiform seizures, and of marked general tremulation.

5. There is also peculiar absence of sensory symptoms, save for blunting of sensibility as the disease progresses.

6. The cases are of long duration, the *average* being four years.

THIRD GROUP.—Principal Changes.

1. The *left* cerebral hemisphere is much more diseased than the right, and is more or less atrophied.

2. There is usually atrophy of the grey cortical substance, most marked in the frontal lobes, but occasionally marked elsewhere. It is usually pale, or pale and mottled by vascular redness, and is sometimes softened, at others indurated in a portion of its extent, either change being much more marked in the left hemisphere, and the frontal lobe being, usually, most affected. The white substance varies in consistence and vascularity.

3. Adhesion and decortication are usually more marked on the left side, occur with equal frequency on the frontal and parietal lobes, while the temporo-sphenoidal suffer very considerably, and the changes in question may be well-marked on the inferior surfaces.

4. The basal ganglia are softened, and their vascularity is altered in either way; as are also the vascularity and the consistence of the pons, medulla oblongata, and cerebellum.

5. The purely meningeal changes are generally well-marked, are either symmetrical or predominate over the left hemisphere, and are often well seen over the base.

Principal Clinical Features.

1. In the stage of mental alteration, preceding that of mental alienation recognized by the friends as such, the patients often are very eccentric, odd, restless, fidgety, and occasionally excited.

2. Dementia, well-marked, early and predominant, is frequent.
3. Melancholic delusions of harm, annoyance, fear, suspicion, are equally frequent, and with them are feelings of alarm and apprehension, or the patients are querulous and irascible, or dejected and weeping.
4. Occasionally there is early maniacal excitement, with irritable passionate outbursts; while exalted delusion, or some largeness of idea, may now and then occur either at an early or at a later period.
5. The later course is generally one of extreme dementia, sometimes with a dash of melancholic, or even of extravagant, delusion.
6. Sometimes destructive, threatening or violent, the patients generally become tractable towards the last, but of degraded habits.
7. Muscular ataxy and paresis are well-marked, motor restlessness is frequent. Finally, the patients are often bedridden, with flexed contraction of limbs.
8. *Hemiplegia* is more or less marked and frequent in all, and is generally epileptic in origin, while more limited temporary palsies, following local spasmodic movements, are frequent.
9. Epileptiform attacks, hemispasm, local spasm are very frequent; and tremor coactus is not unfrequent. Apoplecticiform attacks and aphasia are sometimes observed.
10. Occasionally hallucinations, general obtuseness of sensibility, or local anæsthesia are observed.
11. The average duration is rather short, being about 17 months.

FOURTH GROUP.—*Principal Changes.*

1. Here the morbid lesions are much more marked in the *right* than in the left cerebral hemisphere. The general description of the changes in the left hemisphere in the last group is here transferred to the right, and of the right in the third group to the left in this. The cerebral vascularity is, however, rather greater in this fourth group.
2. The adhesion and decortication are usually more marked in the right hemisphere, occur mostly over the parietal lobe, often on the posterior part of the frontal and on the temporo-sphenoidal, and occasionally upon points of the internal surface or base.
3. The other changes are much as in the third group.

Principal Clinical Features.

1. Occasionally preceded by a history of strangeness and peculiarity of conduct, the outbreak generally begins with ambitious delirium, with or without active maniacal agitation, and violent, destructive and dangerous tendencies. At first there is usually complacency, elation, or exaltation of feeling.
2. Now and then dementia, with fidgety, mischievous, restless, slovenly, or destructive tendencies.
3. Later, there are often exaggerated or exalted notions, alternating with conditions in which the patients are foul-mouthed, querulous, morose, irritable, depressed, or in dread; or the latter states come to predominate entirely.
4. At first there may be the expression of an undiscerning generosity, which is replaced later by an abusive and foul manner of address, often accompanied with degraded habits, and destructive or dangerous tendencies.
5. The muscular ataxy and paresis are of the ordinary type. Occasionally there is great tremulousness, or, again, tremor as in paralysis agitans. Hemiplegia is frequent, sometimes occurring as a simply paralytic seizure, sometimes following epileptiform attacks, and sometimes due to embolism, or hæmorrhage.
6. Epileptiform seizures are very frequent.

7. Sensation is blunted in the later stages; occasionally there are hallucinations of sight and hearing, blindness, or hypochondriacal sensations.

8. The duration is medium, or rather long, the *average* being 24 months.

Thus it will be noticed that some of the clinical features are very dissimilar in the third and fourth groups; mental symptoms like those of dementia and of melancholia predominating when the *left* is the hemisphere principally diseased; and exalted delusion and maniacal agitation when the *right* is the hemisphere in which the morbid process is earlier, and more extensive, severe, persistent, and disorganizing.

FIFTH GROUP.—*Principal Changes.*

1. There is local induration of the cerebral cortex, sometimes of wide distribution in its lesser degrees, most marked in the frontal lobes or their anterior portions, and affecting either one hemisphere or both.

2. The indurated cortical substance, generally of a decided reddish colour, is occasionally pale. It is usually atrophied. The non-indurated cortical substance is of ordinary colour, or pale.

3. The adhesion and decortication, absent in one case, are in others unequal in the two hemispheres, occur mostly on the parietal, often on the posterior part of the frontal, and on the temporo-sphenoidal lobes; now and then on the internal surfaces; and in one case were highly marked on the inferior surface.

4. The white substance, generally of slightly increased consistence, may be fairly vascular, or paler than usual. Generally there are changes in the parts at the base of the brain and in the spinal cord.

5. The purely meningeal changes are well-marked, and of very wide distribution.

Principal Clinical Features.

1. The mental symptoms are various and varying. Some suffer mainly from symptoms of mental depression; others, of dementia; and others, of maniacal agitation and emotional exaltation.

2. Complacency, passionate irascible outbursts, gloom, depression, or apprehension are observed in various cases.

3. All are indifferent to their degraded habits, and some are docile throughout, but others are at some periods either destructive, or quarrelsome and abusive.

4. Muscular ataxy and paresis are fairly marked. At the last the patients are usually bedridden, with flexed contractions of the limbs.

5. Epileptiform fits, hemispasm, often followed by epileptic hemiplegia, are very frequent. Local spasms, followed by more limited incomplete palsies, are not infrequent. Sometimes tremor coactus, or, again, choreiform movements are observed, or apoplectiform seizures, some with, some without convulsive movement. Besides these, several had less grave, but frequent, attacks of heaviness, drowsiness, and semi-stupor.

6. A few show hallucinations of special senses, or marked anæsthesia, or early headache.

7. The *average* duration was 23 months.

A few words may be added as to the *microscopical* appearances in the cerebral cortex in these several groups.

In the *first* group the blood-vessels are distorted, dilated here and there, and their walls the site of degeneration, of deposits, and of nuclear hyperplasia; while interstitially there is hyperplasia of the nuclei of the neuroglia, or proliferation of embryo-plastic nuclei; free blood corpuscles, and many scattered collections of blood-pigment. The nerve-cells are more or less degenerate.

In the *second* group, the nerve-cells suffer perhaps more than the other elements of the cerebral grey cortex. In one case the atrophy and destruction were so marked in the frontal lobes that there seemed to be an absence of the large nerve-cells; the nerve-cells present were comparatively small, atrophied, with rounded wasted outlines, and possessed of but few processes. Some had undergone marked granular degeneration. Some had fallen asunder and the granules, strewn about, looked like a downfallen heap of pebbles. Even the hyperplastic nuclei of the neuroglia had undergone degeneration.

In the *third* group, that in which the *left* cerebral hemisphere is the one much more diseased, the fact is obvious under the microscope as well as the naked eye. Thus, for example, in one case the grey cortex of the third left frontal gyrus showed some granular degeneration of the nerve-cells, hyperplasia of the nuclei of the walls of its blood-vessels, and thickening of the coats of some of the small vessels, of which certain had also dark grey deposits or growths in the walls; increase of neuroglia or free formation of embryoplastic nuclei, and some colloid bodies. Here, as compared with the corresponding part on the right side, the nerve-cells were more granular, there was more increase of the neuroglia and its nuclei, or of "embryoplastic nuclei," and the colloid bodies, present here, were absent in the right side. In the spinal cord were compound granule cells, colloid bodies, and some granular degeneration of some of its nerve-cells.

In the *fifth* group the microscopical indications of interstitial sclerosis are well-marked.

Concerning the above groups of cases, a few words may be added:—

The *first* consists of cases of a very common kind, illustrations of which are abundant in medical literature. Bearing in mind that it represents the shorter and more acute cases of a larger group, it may be passed without further comment.

But not so the *second* group, in relation to which some will, perhaps, raise objection. Possibly some would call these cases atrophy of brain, or chronic hydrocephalus, or chronic meningitis, but an attentive study of the clinical features and necroscopic records will suffice to justify the position in which they are placed here. Bayle fully described cases of this kind, and several of them may be found in his second series. Drs. Baillarger and Lunier distinctly assert a place among general paralytics for cases somewhat similar. But those to be detailed hereafter as composing our second group will be found to approach much nearer to the typical cases of general paralysis than to the hydrocephalic, and other, cases just referred to. In fact, out of a large number of cases, a series could be selected, which, by gentle gradations, would lead the steps from this group up to the most characteristic and typical case of "ambitious monomania, with general incomplete paralysis."

The *third* group, with its less usual symptoms, is relatively infrequent. The symptoms are not at all constant, nor is this to be wondered at, as the principal lesions may be brought about in different ways. Turning to Calmeil's work I find that his cases with lesions somewhat like those of the third group, presented, also, clinical features, on the whole very similar to those of the latter [T. I., pp. 385, 554. T. II., pp. 27, 53, 76, 89].

To the *fourth* group the same general remarks apply. The several cases in Calmeil's work, presenting somewhat similar lesions, were also, on the whole, manifested by very similar symptoms, except that in them attacks of apoplexy and stupor and of spasmodic twitching were more frequently observed.

In the *fifth* group the interstitial changes tend to sclerosis. The pathological process is different in its results from that which produces the more ordinary softening of general paralysis. And this, notwithstanding the view of some, that the pathological process is really the same in the two cases. This group, however, is not so well defined, clinically, as the others, and I do not lay special stress upon it. I find that Calmeil's cases, with somewhat similar lesions, exhibited also a clinical similarity to those of the fifth group, but, as compared with the latter, presented more of violent maniacal excitement, and délire ambitieux, and exaltation; less sadness, less epileptiform convulsion followed by hemiplegia, and somewhat less

spasmodic twitching and tremor cōactus. Otherwise, as in frequency of attacks of apoplectiform nature, somnolence, stupor, the two groups are very similar. [T. I., pp. 311, 431, 437, 519, 571, 581, 591, 658. T. II., pp. 5, 60.]

I am quite aware it may be said that the *mental* differences to which I have referred in the above groups do not mark any essential differences in the cases; that, for example, it may be urged that the grandiose delirium is, in reality, only a manifestation of that dementia which, on the mental side, appears to be of the essence of the affection. Such, indeed, was the view of the elder Foville, Bailarger, Lunier, Jules Falret and Sankey, while that of Guislain, Billod, or Griesinger was not very dissimilar. I am content to record the facts as I find them, believing that differences in the *mental* symptoms of general paralysis, though not essential, are yet of valuable import.

The views expressed in this paper are based simply upon the clinical and necroscopical observation of a number of cases; upon the fidelity of this they must stand or fall; nor need one be concerned to trace a harmony or discord between the facts mentioned above and the conflicting results of experiments on the localization of cerebral function.

CHAPTER XI.

CASES OF GENERAL PARALYSIS.

In order to curtail what follows I have omitted many of the cases upon which my paper was founded, and have abbreviated the clinical and necroscopical descriptions of those recorded here; omitting many, and briefly summarizing others, of the numerous and voluminous notes made in every case. Yet it is trusted that sufficient fullness of detail has been retained, and that the number of cases is adequate, to place those who take any interest in the subject in the same point of view as occupied by the writer.

Particularly has the *first* group been abbreviated, representing as it does comparatively accentuated and rapid cases of the most common form of general paralysis; while only the headings of the cases of the *fifth* group have been finally retained.

FIRST GROUP (see p. 180).

CASE I. (in abstract).—*Severe and protracted maniacal excitement; extreme motor agitation; masking the paretic indications to a considerable degree; ambitious delirium; usually pleasant good-humour and exaltation of feeling; transitory weeping. Pulse rather slow.*

Widely distributed encephalic hyperæmia and softening; extensive adhesion and decorication of grey matter, especially over the supero-lateral fronto-parietal regions and uncinate gyri, and well-marked over the internal surfaces.

J. T. R., a driver in the Royal Horse Artillery. Admitted Feb. 13th, 1874. Age 34. Married. Length of service, 16½ years.

History.—Marked symptoms dated from Dec., 1873. This was the first attack of mental disease, and before coming here he had been treated in the regimental hospital at Birmingham and in the Royal Victoria Hospital at Netley. He was stated to be neither epileptic nor suicidal; and the causes assigned were reported in vague terms as being of both a "moral and physical" nature.

He was reported to have been suffering from general paralysis whilst at Netley,

and to have been in a constant state of active excitement, passing restless and sleepless nights, and exhibiting incoherent irrational language and extravagant delusions. He also said his wife came in through the ventilator, and he tried to stuff it with a pillow. Emaciation steadily progressed. He remained noisy and of wet habits at night, and the restless excitement, the incoherence, and the extravagant ideas as to wealth continued up to the time of his transfer from Netley to Grove Hall Asylum.

State on Admission. Physical Condition.—Height 5 ft. 5 in. Weight 133 lbs. Pulse 72, regular; arteries somewhat tense; no cardiac bruit, or change in area of præcordial dulness. Scar of venereal sore on penis. A few furuncles on body. Slightly florid complexion. Pupils equal and acting fairly well. Gait a little awkward and unsteady, especially in turning. Viscera healthy.

Mental Condition.—He was self-satisfied, smiling, exhilarated, loquacious, irrational and incoherent in conversation. Thus he said "Mrs. S— is my husband, the Lord Bishop." "My rank is about (that of) a Lieutenant-Colonel." "I've plenty of money—all the nation is my money." "My pay is a Bishop's pay—some millions." "I've £700,000,000 in the bank."

Subsequent Progress of Case.—Several days later he was noisy, and of changeable temper, now excited, now angry, now placid for a moment, and, again, weeping like April shower. He said that he "had educated the Queen to manage armies and control the nations." He busied himself in collecting imaginary "droves of bishops." Apparently he had hallucinations of sight and of hearing: for example, becoming excited and refusing to be examined he turned as if gazing at some apparition and said, "See! Major Th—." Then as if repeating the advice given to him by the phantom Major he shouted, "Kick him? Yes I will, I'll do as you tell me." On being questioned he pointed to the Major "dressed in gold," and revealed the delusion that he had seen and spoken to a Mr. G— ten minutes previously. He gave expression to extravagant, absurd, mobile, varying, and self-contradictory delusions as to possession of wealth and power, and often stood talking to the wall, or shouted while capering about and confronting it. There was an occasional slight pause or hesitation in the speech, which was loud and excited, and there was perhaps a faint fibrillary tremor of the tongue on protrusion, but not of the face during speech. Tinct. Digitalis and Potassic Bromide.

The excitement and restlessness were so incessant and extreme that he was emaciating rapidly, although on full diet, and therefore Ol. Morr. and Ferri Perchlor., were also ordered. Yet he was found to have lost 6 lbs. in weight at the end of eighteen days. Then he was ordered to take 1½ lbs. of minced meat daily; also beef-tea and extra bread. After using this food for twenty-five days he had gained 9 lbs. in weight. During this period left othæmatoma had developed, and the patient continued to be restless, loquacious, incoherent and destructive.

In April, noisy and loquacious, he was still in almost constant motion, clattering his feet on the floor, destroying his clothing, being wet by day and dirty at night, and smearing his bedding and the walls of his room with feces. His language continued to be incoherent and expressive of the most extravagant and utterly disconnected notions, and their relics,—words such as "millions" and "bishops" forming the nuclei of a farrago of nonsense that he sang by the hour together with the utmost exaltation of feeling and manner. Weight on April 14, 137½ lbs. After this, Succ. Conii was ordered, but the weight fell to 133 lbs. by May 18th, and then Ext. Phosphostigmatis was substituted for the Conium, and the oil, iron, and extra diet were continued. Mentally, unchanged. The pulse, usually rather slow, became more frequent under conium.

July 27th. Had diarrhoea. The facial and speech signs of general paralysis were still only slight. Was again very restless at night. Sept. 7th. Weight 142 lbs., his highest weight whilst here. Sept. 14th. Had a syncopal seizure this morning. Omitted the Phosphostigma and other remedies.

On Oct. 9th profuse diarrhoea returned, and continued with intestinal hæmorrhage. Both persisted, mental confusion and stupor supervened, these passed into coma, and this into death on Oct. 13th, 1874.

Abstract of Necropsy.—25 hours after death. Body somewhat emaciated.

Head.—Calvarium of slightly worm-eaten appearance along the track of the superior longitudinal and lateral sinuses. A small flattened exostosis on the internal surface of the right side of the frontal bone perforated the dura-mater. Two fluid-ounces of serum escaped on removal of the brain. Arteries at base of brain healthy. The cerebral meninges were injected with blood, especially at posterior part. The gyri were somewhat shrunken, and the slightly wide and rounded sulci were filled with serosity, which bathed the pia-mater on the superior surface except over the occipital lobes. The pia-mater and arachnoid were somewhat thickened, and the former was hyperæmic and separated with difficulty from the cerebrum. There was some opacity of arachnoid over the frontal and parietal lobes.

The superficial layer of cortical grey matter of the prominences of the gyri stripped off along with the membranes over the whole of the superior and lateral aspects of the cerebrum in the fronto-parietal region. Especially was this marked in front, where the entire outer layers of the grey matter stripped off, but every convolution of the area just specified was extensively involved, and further detail is unnecessary. The superficial grey matter came away also from the internal surfaces of the cerebral hemispheres. The same change also affected the prominences of the first and second temporo-sphenoidal gyri to a moderate extent and degree, but tapered off here and spared the third gyrus. The occipital lobe only showed the adhesion-changes, and to a slight extent, in the portions of the first and second occipital gyri which border upon the parietal lobe, or assist in forming the annectant convolutions. The grey matter of the uncinatæ gyri also separated along with the meninges, and a few scattered points of adhesion were found elsewhere on the inferior surface of the brain. The grey matter was hyperæmic, mottled by sections of the contents of visible dilated vessels, slightly softened, of a deep grey and somewhat slaty hue, of fair depth, and of imperfectly marked stratification.

The white substance of the brain was somewhat softened, and was mottled, pinkish, the puncta sanguinea were numerous, and clots dragged therefrom in making sections. All above-mentioned appearances were symmetrical in the two hemispheres. Fornix softened, and serum in lateral ventricles turbid. The left corpus striatum and optic thalamus seemed to be slightly shrunken, but there were no special morbid appearances on section. The pons Varolii and medulla oblongata were lessened in consistence, hyperæmic, and their meninges hypervascular. The cerebellum was hyperæmic and slightly softened. Weight of cerebrum 40ozs., of cerebellum 5½ozs., of pons and medulla oblongata 1oz.

Thorax.—The heart weighed 10½ozs., its left ventricle was moderately contracted, and was one inch at its point of greatest thickness. Its appearance was that formerly called "concentric hypertrophy." The heart-muscle had a healthy appearance. *Right lung*, weight 25ozs.; posterior and basal congestion. *Left lung*, the same; weight 23½ozs.

Abdomen.—Old peritoneal adhesions were found. *Liver*, 80ozs., dark on section. *Spleen*, 10ozs., diffuent, of a dirty brick colour, hobnailed and of irregular shape. *Right kidney*, 10ozs.; *left kidney*, 9ozs.; of variegated appearance, from irregular whitish districts.

Remarks.—1. This is specially interesting as an illustration of the earlier stages of those cases in which the ataxy and paresis are *masked* by the effects of the maniacal excitement. It will be noticed how slight, comparatively, were the ataxic or parietic indications afforded by the condition of speech, tongue, or gait; and yet how well-marked the ambitious delirium and the maniacal agitation. Had the patient lived longer the motor disorder and impairment would have become more and more marked. Such disorder of the motor power as did exist was, perhaps, relatively more marked in the lower extremities than in the upper.

2. This case also illustrates that form of extreme restlessness and motor agitation in general paralysis, that Bayle called "convulsive" and that is exemplified in several cases of his third series. ("Traité des Maladies du Cerveau et de ses Membranes," p. 144.)

3. The rapid emaciation of this patient was arrested only by the use of a very

large amount of nutritious food, with Ol. Morr., and Ferri Perchlor. No means employed had any permanent control over the nearly incessant mental and motor agitation.

4. The heart and arteries were comparatively unaffected by the moderate renal change. The condition of the left ventricle was, at least mainly, due to the mode of death.

5. Adhesion and decortication were very extreme in degree and extent, and the other morbid changes of the encephalon very general.

CASE II.—Extravagant and absurd delusions, with much and early mental weakness. At first, exaltation of feeling; later on the patient alternately self-pleased, dull, and lachrymose. Childish simplicity. Dementia, becoming more marked. Progressive paresis; bedridden; bedsores: hæmorrhagic maculæ. Apoplectiform seizures. Late tremor clactus; and, finally, choreiform movements.

Extensive encephalic hyperæmia and softening; widely-spread adhesion and decortication, especially over the superior and inferior surfaces of the cerebrum, and affecting the cerebellum also. Marked changes—softening and other—in the spinal cord. Granular kidneys.

A. B., Private 100th Regiment. Age 35. Military service 15½ years. Admitted Aug. 2, 1873.

History.—This was stated to be the first attack of mental disease. It had existed since about April, and the patient had been under treatment at Portsmouth, and at Netley. The "cause assigned" was vaguely stated as being of a "physical" nature. The patient was said to be neither epileptic nor suicidal.

The mental symptoms appeared to have come on gradually, and the patient was stated to have had "delusions of a religious type" at first, but at Netley the symptoms were said to be those of "dementia" coexisting with "a marked paralytic tendency," and it was further certified that A. B. was quite confused, restless, incapable of taking care of himself, frequently out of bed at night, and walking about at unseasonable hours, and that he gave indications of impairment of memory, confusion of thought, and failing powers of comprehension and of utterance.

State on admission into Grove Hall Asylum. Height 6ft. 1½in. Well nourished, and well built. Thoracic and other viscera apparently healthy; pulse 96, arteries slightly tense. Pupils equal.

The speech, and the condition of the lips, face and tongue, were those usually observed in the comparatively early or middle periods of general paralysis.

The patient could not tell his own age correctly, mistook the month and year, was confused and incoherent in continued conversation, displayed general mental enfeeblement, and asserted that he was heir to his uncle's estate worth £19,000. His manner was simple and childish, his expression was usually smiling, pleased and satisfied, and he was still clean in person and habits. Ordered Ferri Perchlor.

Oct. 30, 1873. On one occasion recently this patient had an attack of cerebral congestion. Aperient enemata brought away large stools, and the symptoms vanished. Subsequently, he took aperients as required, the iron being continued. Slight "congestive" symptoms had appeared on several other occasions.

Jan. 30, 1874. The tremulous twitchings of the lips and tongue during movement were now well-marked; speech was slow, broken, hesitating, indistinct. The pupils were irregular, the right vertically oval, the left large, and both fixed. The grasping power of the hands was impaired, and the gait unsteady, the left lower extremity was the weaker, the knees and hips were kept partially flexed as he walked, and as he spoke the head shook. He said he was well and strong, and he had exalted delusions as to his possessions, physical prowess, and procreative powers. Yet he was not much elated. On the contrary he was generally dull, confused, and often had an apprehensive, anxious expression. He was often restless and sleepless. Had Ergot and Potass. Bromid.

March 1. In bed with signs of bronchitis and congestion of the lungs since Feb. 18th; there were also præcordial oedema and increased dulness, and the heart-

sounds were heard more distantly. Now, he was often lachrymose, sometimes very dull and confused, at others brighter. He was slow to reply and spoke with very marked tremor and quivering of the lips. There was marked tremor and paralysis agitans (symptomatic) of the forearms and legs, the limbs quivered constantly therewith, and the eyelids were generally closed and quivering. Reflex action was nearly abolished in the feet, but he retained some voluntary power over the lower extremities.

15th. Bedsores had formed over the hips and sacrum, and on the body there were scattered minute hæmorrhagic elevations of lichen-like form. His mutterings now and then revealed the shattered relics of delusions of wealth, and occasionally he sang snatches of song.

24th. Tremor cœactus still marked. Hypostatic pneumonia. 26th. Occasional retention of urine latterly, and drowsiness. Legs œdematous. Rapid flexion movements at the wrist, much tremulous twitching about the arms generally, also about the legs, feet, the head, platysma, and cervical muscles. But the extremities were also affected by conspicuous choreiform movements. Death on March 28, 1874.

Sectio cadaveris; 44 hours after death

Head.—Calvarium thin. Dura-mater hyperæmic. Arachnoid at base rather thickened; frontal interlobar adhesions. The arachnoid was thickened and opaque at the vertex, and the pia-mater thickened, hyperæmic, and its surface bathed in serosity. The anfractuositities, wide and rounded on the upper surface of the cerebrum, afforded a lodgment to serosity, and shreds of the soft meninges remained behind in them after the process of general stripping off. There were marked adhesion and decortication affecting the prominences of all the gyri of the frontal and parietal lobes on their superior and lateral surfaces, also affecting the inferior (orbital) frontal and temporo-sphenoidal surfaces to a less degree. The grey cortical matter of the cerebrum was also hypervascular, pink especially in its deeper layers, somewhat softened, of fairly marked stratification, and moderate thickness. The white substance of the cerebrum was also hyperæmic and softened. The fornix was softened, the velum interpositum tough, the grey commissure small, the lateral ventricles of the brain were large but not containing abundant fluid, the corpora striata somewhat softened, the optic thalami hyperæmic.

The cerebellar meninges, also, separated with difficulty, and carried off with them a superficial layer of the grey substance from the prominences of the folds in the vicinity of the median line. Cerebellum softened. Weights. Cerebrum 39½ozs.; cerebellum 5ozs.; pons V., and med. obl. 1oz. Serum from cranial and spinal cavities, 3½ozs.

Spine.—The meninges of the spinal cord were hyperæmic about the mid-dorsal region. The spinal cord itself had undergone softening in the cervical and upper dorsal regions, and was somewhat anæmic in these parts. The softening was more marked in the cervical region, and there principally affected the grey matter, which was almost diffuent, and the antero-lateral columns.

Thorax.—The heart weighed 10ozs., and was healthy, but there was 1½oz. of pericardial fluid. The aorta was atheromatous. In both lungs there was hypostatic pneumonia.

Abdomen.—Liver of slightly "nutmeg" appearance, 67½ozs. The kidneys weighed, R., 4½ozs.; L., 5½ozs.; their cortices were thin and granular, and their capsules adherent. The middle tract of the ileum was hyperæmic.

Remarks.—1. This was a case of well-marked general paralysis, running a somewhat rapid and severe course. Early dementia mingled prominently with the extravagant delusions; the early self-satisfaction gave place to a mobile and varying state of feeling; locomotor helplessness came on at a somewhat early period, and various trophic lesions supervened.

2. Tremor cœactus, or symptomatic paralysis agitans, and, finally, choreiform movements were superimposed on the marked tremulousness and twitching common in general paralysis. In relation to them we note the unusual amount of disease in the spinal cord and at the base of the brain.

3. There was also an unusual amount of meningeal adhesion, and consequent erosion of the cerebral grey cortex.

CASE III.—*Early exaltation with delusions, auditory hallucinations and restlessness. Later, almost complete remission of the mental symptoms, continuance of the physical (motor). Return of slight ambitious delirium. Self-satisfaction. Almost constant singing and joyous good-humour. Later, restlessness, excitement, destructiveness. Phthisis pulmonalis.*

General softening of encephalon and hyperæmia, the appearance of the latter, however, being diminished by the mode of death. Extensive adhesion and decortication, especially over the superior surface of the frontal convolutions, and the lower tiers of the parietal convolutions. Changes in and about the basal ganglia. Impaired nutrition of the muscular tissue of the heart.

T. H., Drummer, First Battalion, Grenadier Guards, aged 34. Admitted Nov. 11, 1872, after 15½ years' service in the army. Single.

History.—This was the first attack of mental disease, and had existed since June, 1872, and on its account the patient had been previously under treatment in the Guards' Hospital, London, and at Netley. The "supposed cause" was stated to be of a "moral" nature. T. H. had been promoted to the rank of drum-major of the 99th Regiment, but his ideas appeared to be so large and his self-importance so inflated that the colonel of that regiment refused to retain his services. He then became restless at night, and one evening he packed up his kit, declaring that he had orders from God to come to London. Afterwards he accounted for his not remaining in the 99th by asserting that the quarters provided for him were unsuitable. He was admitted at Netley with slightly exalted delusions, and some failure of the powers of utterance. Afterwards he became worse, the delusions more prominent and associated with some excitement and with auditory hallucinations. He also undressed at unreasonable hours.

State on admission here. Height, 5ft. 10in. A fine, soldierly, well-built, handsome man. Well nourished, fresh complexion, pupils rather small, the left slightly the larger, both sluggish and of slightly irregular outline. Skin delicate, healthy. The tongue is protruded fairly, but the aid of the teeth is often invoked to grasp and steady it. There is paresis of right side of mouth, the speech is now and then imperfect, the words being clipped, and there is an occasional pause, with thickness and indistinctness of utterance.

Mental State.—Although he says that he is a clever musician and that he left the 99th because his quarters were unsuitable, yet he denies all his former hallucinations and delusions, and the remission in the mental symptoms of "general paralysis" is decided. He denies having ever indulged in any alcoholic or sexual excess.

The subsequent notes of this case may be briefly summarized as follows:

There gradually supervened a state of more or less mental confusion, now and then accompanied with delusions that his friends were about to come for him, while his general expression and bearing were those of great self-satisfaction and joyous good-humour. The ataxic and parietic symptoms still progressed, and the treatment was with digitalis and iron.

In February and March 1873 there was a gradually increasing excitement, manifested by the reiterated singing of a narrative of various trifling events in his life, and of an enumeration of his possessions, intentions, and good qualities. Together with this monotonous sing-song, carried on for hours together, there were restlessness and a disordered state of his dress. The physical signs of phthisis were observed; then emaciation appeared and was progressive, and due, in part, to the continued restlessness and excitement. He was also destructive. At the end of March he was confined to bed on account of the pulmonary condition, and for the restlessness and sleeplessness calmatives were required. His habits were wet and dirty, he grew very feeble, and tremulous, and died in a syncopal attack on April 16, 1873.

Necropsy (in abstract). 21 hours after death. **Head.**—Cranium thick. About 7½ ozs. of serum, in all, escaped from the cranial and spinal cavities. Interlobar adhesions. The arachnoid over the superior surface of the brain was somewhat thickened, opalescent, and tough. The convolutions of the brain were loosely packed on this aspect, and subarachnoid fluid filled many of the anfractuositities. The superior meningeal veins

were full, and the pia-mater was still hyperæmic. There was a wide-spread adhesion of the membranes to the convolitional grey matter, and consequent partial decortication of the latter on stripping off the meninges. This was especially seen on the superior and lateral surfaces of the cerebrum. The several frontal convolutions, and the lower tiers of the parietal convolutions (asc^d. p., s. margⁱ., ang^r.) were those most markedly affected; the remaining convolutions of this area were less involved. The same adhesion and decortication also affected the cerebellum, whose arachnoid membrane was opaque. The grey matter of the cerebral cortex was diminished in consistence. The white substance of the brain was also universally softened, and was the site of considerable vascularity. The lateral ventricles of the brain were large, and contained loz. of serosity. The fornix was soft, almost diffuent; the corpora striata and optic thalami were soddened and softened; the pons V. was softened and of a pinkish hue; the posterior part, especially, of the medulla oblongata was softened and hyperæmic. Cerebrum, 31½ ozs. Cerebellum, 5½ ozs.; pons and med. obl., loz.

All the viscera were carefully examined, but it is only necessary to state here that both lungs were phthisical,—that the muscular substance of the heart was unduly friable, and of a somewhat pale and yellowish hue, and that a considerable amount of fat was found on the exterior of the heart. Weight of heart 11 ozs. The aorta was studded with patches of incipient atheroma. Spleen 12 ozs., soft, diffuent, of prune-juice colour. Other viscera healthy; kidneys 8, and 7½, ozs.

Remarks.—1. This case is an example of that clinical form of general paralysis in which there is self-satisfaction, joyousness, abundant good-humour, the recounting of trifling events with much gusto, and general childishness and feebleness of mental power, rather than decided ambitious delirium.

2. The hyperæmia of encephalon and meninges was lessened by the mode of death. Adhesion and decortication were wide-spread: the basal changes were well-marked.

CASE IV.—Convulsive seizure, followed by failure of the mental powers, and defective speech, hearing, and locomotor power. The ordinary indications of labial and lingual ataxy and paresis not so well-marked as usual. Extreme dementia and asthenia. Unusually short duration.

General softening and hyperæmia of the encephalon and spinal cord. Widely spread adhesion and decortication of cerebral convolutions. Wasting, especially of the frontal convolutions, and of those in the anterior part of the parietal area.

It is unnecessary to relate the clinical history or more of the necropsy than refers to the brain.

Necropsy: 87 hours after death. Fairly nourished; bleb on right malleolus.

Head.—Calvarium very thick and dense, the portion removed weighing 20 ozs. Dura-mater slightly thickened, and its adherence to calvarium slightly increased. The arteries at the base of the brain were healthy. There were interlobar adhesions. Meninges congested. The visceral arachnoid and the pia-mater were thickened, and the latter was infiltrated with serum in parts; especially over the superior frontal, and anterior part of the superior parietal surfaces, where, also, the anfractuositities were rather wide and rounded.

There was adhesion between the pia-mater and the grey cortical substance,—and corresponding erosions of the latter when the former was removed,—over the frontal convolutions, over the convolutions of the anterior part of the parietal lobe, and to a moderate degree over the temporo-sphenoidal gyri. The superior and lateral surfaces of the first two lobes, the lateral surface of the last, were those principally affected. This change did not involve the occipital lobe, but was found strewn in patches over the internal surfaces of the cerebral hemispheres, and to a moderate degree on their inferior surfaces. It was nearly symmetrical in its disposition on the two hemispheres.

The cortical grey substance of the cerebrum was hyperæmic, of a mottled reddish pink colour, softened, of fair depth; its stratification was imperfectly marked. The white cerebral substance, universally softened, was hyperæmic. Of turbid serosity in

the lateral ventricles there were about three drachms. The grey commissure had disappeared. The corpora striata and optic thalami were softened and hyperæmic. The meninges covering the base of the brain, especially the orbital region, were slightly thickened and opaque. Cerebellum, pons Varolii, and medulla oblongata and upper part of spinal cord were all diminished in consistence. Weights. Right cerebral hemisphere, 19½ozs.; left, 19½ozs. Cerebellum, 5½ozs.; pons, and med. obl., 1oz. Serum from brain, more than a fluid ounce. Granular, and other, renal disease.

GROUP II. (see pp. 180-1).

CASE V.—*Protracted duration. At first, excitement with delusions, succeeded by a somewhat steadily progressing fatuity, with a low and brutish state of feeling. Impairment of speech and of locomotor powers. Patient bedridden for a long period at the last. Sensory impairment. Speech less affected than usual; incessant grinding of teeth and noisy sniffing. Wet and dirty habits. Phthisis pulmonalis, and, finally, spinal tuberculosis.*

Marked atrophy; softening; and some anæmia of brain. Marked meningeal changes. Moderate amount of adhesion of the pia-mater to the cerebral cortex, especially in the neighbourhood of the lower border of the Sylvian fissure. Marked chronic disease of spinal cord, as well as recent spinal tuberculosis.

C. C., Private 56th Regiment. Service, 8 years. Age, 28. Married. Admitted January 24, 1874.

History.—This was stated to be the first attack of mental disease, and to have existed since 1871. He had been under treatment in 1871-2-3 in India, and latterly at Netley for one month. The effects of tropical climate were supposed to have, at least, assisted in causing mental derangement. At the onset of the disease in India he was the subject of delusions with excitement.

At Netley his habits were dirty, he was irrational, hoarded rubbish, and showed passing fits of excitement; his mind was quite prostrate, he was unable to comprehend a simple question properly, and he seemed to know but little except his own name.

State on Admission.—Height, 5ft. 4in. Weight, 148lbs. Well nourished. Sallow aspect. The pulse was 66; the pupils were equal and sluggish. The tongue was a little tremulous, the speech thick and hesitating, and dwelling as it were upon the words. He had a peculiar habit of loudly sucking his lips, which were livid. The gait was unsteady, and he wetted the bed at night.

His mind was a wreck; but little response could be elicited from him, and when elicited his replies were irrational and incoherent. He was dull, apathetic, and exhibited neither gaiety nor sadness, neither interest nor aversion. R. Ferri Perchlor.

Shortly after his admission the habits became more constantly wet and dirty, and othæmatomata appeared.

Early in 1875 he remained in the same unintelligent condition; to questions replying either not at all, or by inarticulate sounds, or by such words or phrases as, "Yes," "I have forgotten." To the former loud "sucking" sound made by him there was now added a frequent and disagreeable loud "sniffing" sound. Physical signs of phthisis pulmonalis.

7th. Pulse 82. Respⁿ. 24. Temperature 100.3°. Phthisis advancing. Add Ol. Morrhuæ to Mixt. Ferri et Quass. It is unnecessary to follow the records as to the pulmonary signs. Indications of pleuritic effusion appeared on the right side. The profuse sweating was relieved to some extent by nightly draughts of Belladonna. The cough was dry.

Grinding of the teeth was frequent, and the habit of "sniffing" most persistent. He was for a long time bedridden, and rarely spoke. Speech though slow, impaired, and somewhat tremulous, was not affected so much as is usual in the stage at which dementia becomes so extreme as here, for he continued to be fatuous, and extremely

and constantly wet and dirty. The expression, which at first was that of hebetude, became latterly rather that of apprehensiveness and fear.

Oct. 3. Pulmonary excavation. Coughing, teeth grinding, and night-sweats, continued. The decubitus was alternately dorsal and dextral.

Oct. 11. Worse for some days. Pleurisy of the left side and pneumonic patches. Vomiting was troublesome, but ceased on Oct. 12th, returned on the 13th, subsided on the 14th. On the 12th the face was flushed, the temperature raised, respiration laboured and accompanied by moaning and an expression indicative of distress.

Oct. 22. Since the preceding day he had been heavy, dull, drowsy. There had been increasing subsultus of the hands and arms, tremors of the lips and face, and marked tremulousness of the movements, as he restlessly pulled the clothes about. The expression was more than usually apprehensive. Pulse 100, weak.

Oct. 23. During the preceding night and until 5 A.M., he took food, but at 7 A.M., was observed by the attendant to be comatose, and unable to swallow, and these symptoms continued. At 9.30 A.M. the pulse was 90, full, quick, compressible, but not decidedly soft. The respiration varied from 44 to 54 per minute, it was irregular in rhythm, in depth, and in frequency, from one half minute to another. Thus, perhaps, at first there were a few audible respirations with guttural sounds, then came a few quiet, easy, noiseless, and less frequent, respirations, during which the heart could be heard distinctly, and its action was then of moderate strength, but its sounds still were feeble and short. There was, therefore, a tendency to "respiration of ascending and descending rhythm." The surface of the body was moist, clammy, flabby, relaxed. The temperature in the right axilla was $95^{\circ}2'$; in the left axilla it was below 95° , (the graduation began at that point.) The conjunctivæ were suffused, watery, insensitive to touch; the pupils equal, dilated, immobile; and the eyes sometimes turned a little to one side or the other. The limbs were quite flaccid, limp, and unresisting to passive motion. There was no distortion of the face. Saliva ran from either side of the mouth that happened to be the lower. A slight, suffused, flush relieved the lately usual pallor of the face. The patient was quite comatose. Later, there was much mucous bubbling in the throat. At 1 P.M. the pulse was 96, weaker and softer than before; the respiration 36 and of irregular rhythm, as in the morning; the temperature below 95° in both axillæ. The left pupil was rather the larger, both were insensitive to light and moderately dilated, the surface of the ocular globe being also insensitive and not reacting to touch. The coma was complete; the eyes and head turned somewhat to the left side now. The face was pale and slightly livid. All the limbs remained equally flaccid, relaxed, and motionless, and the inability to swallow, and the rattling of bronchial mucus were as before. The moist and relaxed skin remained cool throughout. Died, same day, at 6 P.M., Oct. 23, 1875.

Necropsy, 73 hours after death. Fairly nourished; rigor mortis.

Head.—Calvarium small, thick, dense. A slightly worm-eaten appearance of its inner surface along the posterior two-thirds of the longitudinal sinus and of the middle fossæ at the base of the skull. Dura-mater very slightly thickened. Considerable serum drained away from the arachnoid cavity, and the base of the skull. There were firm interlobar adhesions of the cerebral meninges. The arachnoid covering the base of the brain was thickened, tough, and of a diffused milky opacity, and in the same region the pia-mater was infiltrated with serum except where the occipital lobe is opposed to the cerebellum. *Right cerebral hemisphere*. The membranes over the superior, lateral, and internal, surfaces of the right cerebral hemisphere were thick, tough, and slightly opaque. The pia-mater was everywhere oedematous, and fluid filled the anfractuositæ, except at the tip of the occipital lobe at the part overlying the cerebellum. This serous infiltration was well-marked on the internal surfaces and was most extreme in the frontal and parietal regions. Adhesion between the cortex and pia-mater, and erosion of the former upon removal of the meninges were especially marked in the posterior part of the frontal, and the anterior part of the parietal, regions; were considerable over both the lateral and the inferior surfaces of the temporo-sphenoidal lobes, as well as over the gyrus fornicatus, existed slightly, also, over the præcuneus and cuneus,

and over the posterior part of the orbital gyri, but were not observed over the tip of the frontal lobe. The separation of the grey substance was from the prominences of the gyri only. The convolutions were nearly everywhere more or less wasted, especially on the superior and lateral surfaces, and in fact were considerably atrophied, except at the tip of the occipital lobe. The grey cortical substance was thin, rather soft, its strata not well-marked, its colour somewhat ordinary. The anfractuositities were very shallow. The white matter was universally softened, and the whole brain was flabby. Enlargement of the lateral ventricles was obvious. The general condition of the meninges was alike on the two sides, and the distribution of the adhesions and cortical erosions was also much the same, except that the left temporo-sphenoidal lobe was less involved thereby than the right, and that little, or nothing, of these changes was seen on the internal surface of the left hemisphere. The white substance was alike on the two sides, both as to softening and hue, being of a mottled faintly lilac colour, and presenting a few puncta cruenta. The fornix was softened, and in parts semi-diffuent. The corpora striata and optic thalami, especially the former, were atrophied, softened, and paler than usual, and were alike on the two sides.

The pons Varolii and medulla oblongata, softened and pale, broke down too readily under pressure. The membranes were somewhat too adherent to the medulla oblongata, and were rather thickened over it. The cerebellum, also rather soft and pale, was covered by opaque arachnoid membrane. Weights. Right hemisphere, 12½ozs. Left 13ozs. Cerebellum, 5ozs.; pons and med. obl., 1oz. Serum from cranial cavity 4 fl. ozs.

The spinal cord was universally anæmic and exhibited a tendency to softening, especially in the lower dorsal region. Strewn all over the internal surface of the spinal dura-arachnoid were beautiful transparent, minute, grey granulations of homogeneous appearance, and tubercular in nature. (Spinal tubercular meningitis.)

Portions taken from the cord did not harden well in the chromic acid solution. Then they stained badly with carmine, and under the microscope there were found in them molecular debris, compound granule masses, fatty molecules, thick-walled vessels, granular nerve-cells, and altered and apparently misshapen nerve fibres.

Thorax.—The heart weighed 6½ozs. Its muscular substance was only a little soft and friable. A few points of incipient atheroma existed in the aortic arch.

Lungs.—Left, 19ozs.; old adhesions, puckered cicatrices, and a vomica at apex. Recent pleurisy with effusion. Recently formed caseous patches in lung.

Over the right lung were old, strong, and close pleuritic adhesions. It contained cheesy masses, with surrounding hyperæmia and tubercular granulations.

Abdomen.—Right kidney 5½ozs.; ordinary cystic degeneration: Left 4½ozs. The spleen was firm, 9½ozs. Liver, 54½ozs.

Remarks.—1. Taking into consideration the whole of the mental and physical symptoms, and the post-mortem appearances, the last being interpreted in the light of the protracted duration of the case, one may conclude that this was an undoubted case of general paralysis.

2. The marked chronic disease of the spinal cord was of interest, as for example in relation to the excessively wet and dirty habits of the patient during so long a period. At first dependent upon intellectual failure, decline of sensory power and of moral sensibility, they were latterly, I take it, dependent in part upon paralysis of the sphincters from spinal disease.

3. In relation to the spinal tubercular meningitis, and the softening of the cord, especially in the lower dorsal region, we find that the closing scenes of life constituted the following drama:—

(a). Death after twelve hours of complete coma supervening somewhat suddenly.

(b). During this period total relaxation and flaccidity of the muscular system, without the slightest convulsion, spasm, rigidity, distortion, or appearance of local palsy predominating anywhere, the condition being a general spinal paralysis.

(c). A low temperature; and moist, relaxed, flabby, pale skin.

(d). The frequent respiration, varying in rhythm, in frequency, in depth, and in loudness, equivalent to a modified respiration of ascending and descending rhythm;

and the coexisting variation of the pulse apparently dependent upon the respiratory variations.

Whether these symptoms and signs were in part due to an extension of the tubercular formations, to the meninges of the medulla oblongata and pons, or even to the base of the brain, and not yet perceptible to the naked eye, is matter of doubt.

CASE VI.—*General paralysis of long duration, about five years or more. At first the physical signs of general paralysis with exalted delusions, the patient being quiet as a rule, but subject to fits of gusty excitement. Then, afterwards, a state of quiet dementia, dulness, and inapprehension, then hypochondriacal symptoms, refusal of food, and emotional agitation. Then, later, occasional paroxysms in which fear predominated; and, lastly, greater dementia often with an expression of confusion, anxiety, or even distress; paresis of all the movements, especially of those of articulation; and, finally, great failure in the power of deglutition, and destructive pneumonia, supervening on tubercular formations. On a number of occasions the right side of the body was slightly more parietic than the left.*

Cerebral meninges thickened, opaque, adematous. Adhesion and decortication slight, and mainly of the second and third temporo-sphenoidal gyri. Atrophy of brain. Moderate wasting of the cortex, which was fairly vascular, and slightly firm. A large amount of intra-cranial serum. Enlarged cerebral ventricles. The hemispheres, including the basal ganglia, were alike on the two sides.

W. T., Private 9th Lancers. Single. Service 13½ years; admitted April 27, 1872, then aged 32 years.

History.—This attack of mental disease was stated to be the first, and to be of uncertain duration, and insidious in its onset. He had previously been under treatment for it at Aldershot, and for four weeks at Netley. The cause was stated to be unknown.

Whilst at Netley he exhibited exalted delusions, and gave expression to great and chimerical schemes for doing universal good. He was irrational and, occasionally, incoherent in conversation, and was liable to gusty fits of excitement.

State on admission.—Physical condition. Height 5ft. 8½in., fairly nourished, viscera healthy, equal pupils. Tongue pale, flabby, a little tremulous; it was protruded slightly towards the right side, and he had a habit of very frequently licking his lips with it. At times, during speech or during protrusion of the tongue tremors or slight twitchings of the muscles of the lips or face were observed. At times the speech was hesitating and indistinct. There was some awkwardness and impaired co-ordination of movement when he turned round, or walked upon a straight line drawn upon the floor. The power of grasping with the hands was good, and he was able to write his name, though not without hesitation.

Mental condition.—The expression was dull, and indicative of mental failure. At times there was a feeling of placid self-satisfaction, but no decided exaltation was evident. His real age being thirty-two years, he stated in examination that he was only twenty-four years old, that he had served in the army for fourteen years after enlisting at the age of twenty-two, that he had been four months at Netley (really one), that three hundred persons came here with him (really ten), that he could do anything, indeed was quite an athlete, that he gave away sovereigns to any one who cared for them, and that he had "£4,000 or £5,000 in the bank," adding the words "more than that—any amount in fact."

During 1872 he betook himself to ward-duties, and for a long time afterwards continued to be industrious, quiet, answering all simple questions in a rational and coherent manner; but never asking to be allowed to leave the asylum, never giving any trouble, and yet exhibiting much more intelligence than he did when first admitted. But the expression still was one of impaired mental power; negative as to emotion, it never evinced depression, and scarcely ever was smiling. Slow and deliberate in all his movements, and apparently of sluggish perception and apprehen-

nion, he was easily put to mental confusion on being questioned. In this condition he remained until the end of 1874.

Jan. 2nd, 1875. He refused to take food and remained in bed; when forced to take food he became very restless, jerky and tremulous, and voluntary movement was attended with convulsive jerks. He declared that "his throat was bad"—"he had no swallow"—"was dying," and he made noises like a wild beast. He drank some milk under the threat that otherwise the stomach-pump would be used, and in drinking swore, and thrust his jaws into the mug and fluid at each mouthful he took like certain of the lower animals. The costive bowels were soon relieved by the enemata ordered. Pupils equal and acting.

Jan. 4th. The patient had subsultus and twitchings of the upper extremities. He would scarcely take any food. The face was somewhat injected; the pulse 81, full and throbbing. The left pupil was now the larger and was irregular. The tongue was furred and the breath rather foul. Speech was much impaired, and during it there were facial and labial tremors and twitchings.

5th. Stomach-pump.

Jan. 8th. He was bellowing, moaning, crying out, "Oh! oh! dear," and anon making strange inarticulate noises. The face was trembling violently, the eyes being kept closed; and the muscles of the trunk and of the limbs were jerked. When the mouth was opened the tongue was seen rolling about, jerked hither and thither almost choreically, but voluntary efforts restrained it. The pulse was full, about 110, but varying in frequency; the face was somewhat injected. He kept muttering incoherently something about "poison," "wrong poison," "wrong dead poison;" "Oh dear—oh dear—the brute—work broken," and so on. On another day he said his teeth were all gone (a delusion).

11th. Pulse 84, respiration 20, temperature 101.2°, the face less flushed than during several preceding days; diarrhoea had continued for several days, and there was a dirty moist fur on the tongue. There was still great tremulousness of the face, tongue, and hands, even when at rest *quoad* voluntary movement, the right hand appearing almost as if affected with slight paralysis agitans. The facial and lingual tremor, and the indistinctness of speech were well-marked. The pupils were rather small, slightly irregular, fairly susceptible; the left was the larger.

April 7, 1875. The pulse was 84 and soft; for many weeks it had varied from 80 to 96. He had been very incapable and bedridden for some time, but had now been up again for a week.

April 13th. Weight 126lbs.

June 6, 1875. He now talked in a somewhat simple and confused way about his past life, and his memory was bad. The habits, which had become dirty, were now improved. The fingers were extremely tremulous in accomplishing any little act, such as the buttoning of his clothes.

August 1st, 1875. The tongue was protruded slightly towards the right side, the tongue and face were tremulous when in movement, the tremulousness was more marked on the right side of the face, and the speech was characteristic of general paralysis. As usual the patient was quiet, and wore a somewhat dull appearance which never became elated. The manner was childish.

Oct. 17, 1875. The signs of general paralysis evinced in the speech and gait were well-marked. Left pupil the larger.

Oct. 28. Weight 152lbs. The increase of weight in six-and-a-half months was 26lbs. This occurred whilst he took extra diet and physostigma.

There was indication of very slightly greater paresis on the right side of the body. During exercise the left pupil grew a little larger, and the right more than doubled its previous size.

The characteristic twitchings and speech of general paralysis were present. There were faint indications of greater paresis of the right side of the face than of the left. He was quiet, and disposed to be hypochondriacal, and to say that his medicine did him harm, that he had lost flesh, and was weak.

April 6th, 1876. He had not been so well just lately, and on the day before he had been extremely dull and stupid, and, the attendant said, "had dragged one

foot." He now walked very unsteadily, the facial and labial muscles trembled very much during speech; or even at other times. Speech was shaky, tremulous, hesitating, and indistinct. The left pupil was the larger, the right was contracted, both were sluggish. The paresis was more marked in the right side of the face and in the right lower extremity than in the left. Face rather pale, pulse full, soft, 100. Weight, 145lbs.; the loss in weight during five-and-a-half months being 7lbs. To omit the physostigma, and take perchloride of iron and quassia.

August 3, 1876. Though very shaky he still walked about a little in the airing ground. The mumbling of speech, the tremors of the face and other parts had increased greatly, and his gait was slow. The right pupil was still small, the left much larger. He was dull, heavy, amnesic, demented, and had a confused anxious distressed look at times. He could not name his regiment, or give his age, or state how long a time he had been here. At times he was most obstinate.

Aug. 8th. He became violently excited when brought to be examined, struggling to escape, and shouting in terror at some product of his own imagination, or of sensorial disorder. But he was easily pacified.

Nov. 3, 1876. Summary—Bedridden and pneumonic as 6th. The patient was ordered to remain in bed, being feeble, helpless, and almost unable to speak or to swallow, and the subject of pneumonia.

6th. Pulse 96, soft; respiration 38; temperature 102.2°. Signs of patches of pneumonia and destructive changes scattered through both lungs.

The difficulty in swallowing remained extreme, fluids tended very strongly to pass into the larynx, and thence into the lungs. If raised up or induced to attempt to swallow, he became much perturbed in mind and made violent respiratory efforts and guttural sounds. Any passing of the cesophageal tube in order to feed him almost produced asphyxia, and only enemata could be resorted to. The pulmonary signs advanced, and he finally died, exhausted, on Nov. 11, 1876; but before decided emaciation was present, or dementia so very extreme as in some cases. And he died mainly in consequence of lung-lesions.

Necropsy.—54 hours after death.

Head.—Calvarium unsymmetrical. Large amount of serum at the base of the brain. Dura-mater slightly thickened. The portion of calvarium removed weighed 17½ozs.

There was a general faint milky opacity of the arachnoid over the base of the cerebrum and cerebellum. Over the whole of the superior and lateral surfaces of the cerebrum the pia-mater and arachnoid were thickened and tough, the pia-mater being also infiltrated with serum, and the arachnoid presenting a white opacity. These changes invaded the membranes covering the occipital lobes, terminating upon them, and leaving the extreme tips unaltered. They were about equally well-marked upon the frontal and parietal lobes, but were much less marked on the temporo-sphenoidal lobes, and were symmetrically disposed over the two hemispheres. The membranes stripped off readily, but left shreds in some of the sulci. The meningeal veins were full, especially behind.

The only points of morbid adhesion of the meninges to the cortex were scattered over the temporo-sphenoidal lobes on both sides, especially over their second and third convolutions, were slightly more marked on the right side, and there affected the inferior surface particularly.

The grey cortical substance was of a pinkish lilac colour, especially its deeper layers; the distribution of this colour was not uniform, there being scattered, small, darker patches, and the naked eye vascularity being greater in the anterior than in the posterior regions of the cortex. The grey matter was, if anything, rather firmer than usual, and of fair depth.

The lateral ventricles of the brain were dilated, and contained a large quantity of serum. Their ependyma was thick and opaque, and the foramen of Monro was widely dilated. The white substance of the brain was highly vascular, its colour deeper than normal, and its consistence was rather firm.

The basal ganglia were alike in the two hemispheres, and the grey commissure was present. The pons Varolii and medulla oblongata were of a pinkish lilac hue.

The lining membrane of the fourth ventricle was thickened and opaque, and the subjacent tissue of a dull lilac colour. Each cerebral hemisphere weighed 17½ ozs., the cerebellum weighed 5½ ozs. and the pons and medulla oblongata 4 ozs. Blood-tinged serum to the amount of 8 fl. ozs. escaped during the removal and dissection of the brain.

Thorax.—*Heart*, weighed 10½ ozs. The muscular substance of the heart slightly friable and darkish.

Right lung, weight 42 ozs., cheesy masses and granulations and destructive pneumonia in patches.

Left lung, weight 34½ ozs., similar changes less advanced, puckered cicatrices in front, small vomices at apex.

Abdomen.—*Spleen* rather firm, of a chocolate hue, 4½ ozs. Left kidney, 4½ ozs.; right ditto, 5 ozs.; both healthy. Liver rather pale, 48 ozs.

Remarks.—1. In this case we find on the one hand a long duration and an unusual course of the disease; (a) the more ordinary mental symptoms of general paralysis, succeeded by (b) quiet dementia, and this by (c) hypochondriacal delirium, and, finally, merging into (d) a state of persistent dementia, with total absence of exaltation, but with the presence, rather, of an appearance of confusion, anxiety, or even distress. And on the other hand we observe a generally wasted and a water-logged brain, together with marked changes in the cerebral meninges, but an absence of meningeal adhesion and cortical erosion, except to a slight extent on the temporo-sphenoidal lobes.

2. Here, also, there was marked motor failure towards the close of life, articulation and deglutition being much impaired, and in consequence of the latter, inhalation of some food and destructive pneumonia took place, and death occurred before emaciation was decided, or the fourth stage, that of amentia, had set in. In relation thereto may be mentioned the somewhat atrophic, yet vascular, condition of the pons Varolii and medulla oblongata, and the marked thickening of the lining membrane of the fourth ventricle and evident subjacent change.

CASE VII.—*Early delusions as to possession of wealth, and as to physical prowess, desultory conversation, self-complacency. Later, the patient confused at times, excited, and subject to transient fits of anger. Still later, he grew more confused and demented, ceased to do any work, suffered much and for a long period from insomnia, and was restless and noisy at night: also suffered from considerable emotional disturbance, and became secretive, obstinate, and excessively coarse, foul and abusive in language. Still later, the patient resisted every manipulation, and howled and roared if touched. Finally, he continued to be utterly repugnant to any interference, dull, stupid, obstinate, morose and peevish, and thoroughly brutish in appearance and action. For years of wet and dirty habits. At first facial twitching and tremor, great motor restlessness, and fidgetiness; and finally, general helplessness and a bedridden state, but the ataxy and paresis of comparatively slight or moderate development during the greater part of the course of the disease.*

The changes of the cerebral meninges very marked and very extensive, and well seen at the base. Great atrophy of brain, large ventricles, and much intra-cranial serum. Cerebral cortex pale; also atrophied, especially in the frontal lobes, and perhaps slightly more so in the right frontal. Adhesion and decortication of grey substance, almost limited to the parts bordering on the Sylvian fissures, slightly more on left side, especially seen on 3rd frontal and 1st and 2nd temporo-sphenoidal gyri, very slightly on the inferior frontal and temporo-sphenoidal surfaces. Some softening and pallor of white substance, basal ganglia, cerebellum, pons and medulla oblongata; and the spinal cord atrophied and somewhat softened. Ulcerative aortic valvulitis, embolism of kidneys and spleen. (Some doubtful indications of internal syphilis.)

J. M., Private A. S. Corps. Admitted June 15, 1872, then aged 32, married.

History.—This was the first attack of mental disease and was said to have existed since January, 1872, and for it he had previously been under treatment at Woolwich

and at Netley, and had been a patient at the latter place since May 7. Mental disease came on insidiously, and by the time he arrived at Netley the existence of "general paralysis" was suspected, and whilst there he was the subject of delusions as to his being the possessor of considerable sums of money, with a general exhibition of self-complacency. His conversation was said to be desultory and irrational, and he suffered from transient fits of excitement.

State on admission.—Height 5ft. 6in., well nourished, pupils equal, clean in person and habits. All the viscera of the thorax and abdomen appeared to be healthy. There were tremors of the facial muscles during speech, which was impaired and ataxic.

He manifested frequent but transient emotional disturbance, especially when spoken to. He believed himself to be, physically, a very powerful man, and said he could lift half a ton weight. Usually self-complacent—often smiling—pleased at trifles, he yet was easily roused to become angry and abusive. Some disposition to occupy himself was shown, and at times his ideas were evidently somewhat confused.

Sept. 1872. But little change had occurred since his admission. Occasionally he was slightly excited, but these attacks were transient. He sometimes assisted a little in ward duties. It will suffice to transcribe a portion of the notes.

March, 1873. For several months he had been slowly retrograding, his ideas being now more confused and limited. He had ceased to occupy himself in any way, and had become destructive to his clothing. His articulation and locomotion were more affected than previously. He had now for some time been taking Ferri Perchlor.

May 22, 1873. Omit the mixture of iron and quassia.

June, 1873. He was becoming worse, and was still very destructive to clothing, but denied his destructiveness in the coolest and most unflinching way.

Dec. 27, 1873. Resumed the mixture of perchloride of iron and quassia.

Jan. 1874. Othematoma of the left ear appeared.

To summarize the notes, it may be stated that during the same year (1874) he was restless, untidy, and from time to time was excessively destructive to clothing. During the second half of that year, however, he was much less disposed to insomnia, restlessness, and noisiness at night, than he had been during the first half of the year. In consequence of the insomnia, and other symptoms mentioned he had taken a night-draught of chloral hydrate from March 20th to April, and of potassic bromide and hyoscyamus from April 15th to July 16th. The iron mixture was continued throughout the year.

Feb. 1875. Furuncle of the left buttock terminating in abscess. In March, chloral hydrate was again ordered for insomnia. On April 25th the use of the mixture of iron, temporarily omitted, was resumed. In May, 1875, oedema of the feet and ankles was present, and increased towards the evenings. The urine contained no albumen and was healthy. An ulcer appeared on the leg and healed under treatment, but afterwards ulcers again appeared, and were aggravated by his persistent habit of picking at them, and the legs continued to be oedematous. He was ordered to bed, and eventually the ulcers healed.

Sept. 29, 1875. The patient was very demented and stupid; when interfered with in any way he shouted and made uproarious complaints. He was still confined to bed, and he still was restless, mischievous and dirty. Oct. 8th. Ordered to get up. Oct. 28th. Weight 149lbs. The patient occasionally fell; he often stood still with drooping figure and head bent towards the ground. The dementia was great. He screamed when he was washed or dressed or in any way attended to, calling out lustily and in a loud and distinct voice, a sentence often being uttered without hesitation or tremulousness. Yet much of his ordinary speech was impaired, and at times was somewhat hesitating, tremulous, and as if stammering, but there were no marked facial or lingual tremors during speech. He was utterly careless, and had a fatuous look, his hair was dry and bristly, his skin rough and coarse, and his general appearance brutish. He would not grasp one's hand, or seize anything when requested to do so, but it was said that his power of grasping was good. Both by day and night he was very wet and dirty. With difficulty he buttoned his coat; often he pulled off his clothes or tore them. At night he was restless, talking

aloud, or pulling the bedding about, and upsetting his utensil. The right pupil was the larger, both were sluggish. The gait was slow, irregular, swerving; the steps were short; the equilibrium was uncertain.

He was irritable, suspicious, and obstinate if interfered with. He said that he had seen his wife the day before, and that she was working here in the laundry (quite erroneous). When questioned, or urged to do anything, however simple, he called out irritably and angrily. When left untouched and unspoken to he seemed to be utterly apathetic, and would stand or sit for hours in one place, if not moved. Often he declared he had nothing to eat, and this delusion seemed to act upon him as any interference or manipulation did, for after expressing it he would swear, call out, or shout aloud. The above condition persisted during the remaining portion of 1875.

On Nov. 30 it was noted that the gait was much impaired; he staggered, and the legs straddled far apart. If not supported he would have fallen every moment, though now and then he could run off for a few steps. When he shouted the utterance was distinct, and tremors were absent or slight. But speech became mumbling at times, and in opening the mouth widely the upper lip trembled much. Memory was greatly impaired, and he was in his usual obstinate, irritable, restless, and mischievous state. On Dec. 25 he was again ordered to bed, where he remained until his death, constantly becoming wet and dirty, and affected with ill-conditioned boils and bedsores.

Feb. 26. Some hypostatic pneumonia of both lungs, especially of the right, for two days, with fever and restlessness: brandy and ammon. carb.; pulse now very soft, very rapid, becoming filiform and imperceptible; respiration 38, moaning, noisy and laboured; vomiting, semi-coma. There was general dull-lividness, with some flushing of the face, and deglutition was much impaired. The right eyelid and pupil were insensible, the right cheek flapped in respiration, and the movements of the facial muscles were mainly confined to the left side. The skin was moist. Vomiting was relieved by several minute doses of ipecacuan and morphia. He died at 2 A.M. the next morning, Feb. 27th, 1876.

Necropsy.—41 hours after death. An unusually foul odour proceeded from the cadaver. Body fairly nourished, considerable rigor mortis, and moderate hypostatic lividity. Small bedsores on hips and buttocks.

Head.—Scalp thick, calvarium thick, especially the frontal bone; portion removed—16½ozs., diploe fairly well-marked. Grooves for meningeal arteries deep and rough. The dura-mater was thick and dense; the pia-mater and arachnoid were thickened and slightly opaque, and these changes were well-marked at the base of the brain, where also there were interlobar adhesions, and over the cerebellum. On the superior and lateral surfaces of the cerebrum the meninges were more than elsewhere thickened, opaque, pale, and highly cedematous, and these changes were about equally marked over the frontal, parietal, and temporo-sphenoidal lobes. The anfractuosités were rounded, and shreds of the membranes were left in their depths. The brain generally was flabby, sinking under its own weight.

Limited adhesion and decortication of grey matter were found; almost entirely confined to the region of the Sylvian fissures, rather more marked on the left side, and mainly involving the third frontal and first and second temporo-sphenoidal gyri. A few isolated patches of this morbid change also affected all the other convolutions which border on the Sylvian fissures. A few slight points of the same were seen on the orbital and inferior temporo-sphenoidal surfaces, but the surface overlying the tentorium was quite unaffected thereby.

The grey cortical matter was thin and very pale, especially the external layers. The pallor was greatest in the frontal and in the occipital regions, the diminution of depth was more marked in the frontal region, and this cortical atrophy was perhaps slightly greater in the right than in the left frontal lobe. The consistence was fair; very few vessels were visible to the naked eye, and the stratification was not very obvious.

The white matter was of a pale faintly pinkish tinge, and was universally soft and flabby.

The lateral ventricles were very large and contained about six drachms of fluid. The corpora striata and optic thalami were of diminished consistence and were alike on the two sides. Right hemisphere $15\frac{1}{2}$ ozs.; left $16\frac{1}{2}$ ozs.

Both the grey and the white matter of the *cerebellum* were pale and of very slightly diminished consistence. Its weight was $5\frac{1}{2}$ ozs.

The pons Varolii and medulla oblongata weighed $\frac{1}{2}$ oz., were slightly pale and of slightly diminished consistence. About 7 fl. ozs. of serum escaped from the cranial cavity and brain.

The spinal cord appeared to be somewhat atrophied, and, if anything, its consistence was lessened.

Microscopical examination, later. In the third left frontal gyrus (a) the larger pyramidal nerve-cells seemed to be singularly absent: (b) the smaller nerve-cells were some of them granular, rounded, with wasted outlines; possessing but few branches, and some had quite broken down, and the granules were strewn about. In parts the neuroglia appeared to be increased. The nuclei of the neuroglia were very numerous, and even some of these had a granular appearance. Large oval granular cells were also seen.

Thorax.—*Heart.* Slight thickening and some opaque patches in the mitral valve. A double perforating ulcer was seen on one of the aortic flaps, and some dirty whitish clot was lying in the sinus of Valsalva behind this. The aortic sinuses were atheromatous. The inner surface of the arch of the aorta was extremely irregular, and presented numerous elevations. They were more or less translucent, and gave a slightly ridged appearance to the interior. The endarteritis deformans was decided. Numerous opaque whitish and yellowish patches were seen in the left coronary artery, as well as a number of superficial calcareous plates, and in the walls of the right coronary artery a firm yellow nodule was imbedded. The weight of the heart, with three inches of the aorta, was $14\frac{1}{2}$ ozs. Its muscular substance was pale, flabby, and friable. Under the microscope granulo-fatty degeneration of the muscular elements was seen.

Lungs.—More or less hypostatic congestion and pneumonia of both. Left lung, 25ozs. Right lung, $24\frac{1}{2}$ ozs.; old pleuritic adhesions.

Kidneys.—The right kidney weighed $6\frac{1}{2}$ ozs., was pale, especially in the cortex, and soft and flabby. The capsule was adherent at only one point. On the surface were numerous minute whitish portions of about the size of a pin's head, each surrounded by a faint pink zone, and scattered in a somewhat regular manner over the surface. Two or three wedge-shaped portions of the cortical tissue, which were homogeneous, of a dirty yellowish white colour, disposed with their bases at the periphery, were also surrounded by a pink zone. The left kidney was the seat of the same changes, embolic and other, as the right. There was also on its surface a small depressed fibrous cicatrix extending a third of an inch into the tissue.

Liver, flabby, pale, rather too friable, $63\frac{1}{2}$ ozs.

Spleen 14ozs., on its external surface was a depressed cicatrix, with slight fibrous bands extending therefrom three-fourths of an inch into the splenic pulp, and ending in a hard yellow nodule, the central parts of which were found to be composed of firm dry white material. Among the constituents observed under the microscope were yellow irregular masses, and cells, containing dark granules, in a reticular tissue, also scattered molecular debris. The spleen was softened and almost diffused, and was generally rather pale, except for one portion of a deep purplish colour.

Remarks.—1. There was evidence of profound change in the blood at the close of life. The odour of decomposition from the cadaver was premature and peculiarly strong, the viscera were soft and flabby, perforating ulcerative endocarditis affected the aortic valves, the clots in the heart were of a dirty whitish-grey colour, soft, friable, and looking as if rotted, numerous emboli were strewn through the kidneys, and embolism of the spleen had occurred and perhaps in the lungs also. These may be examined in a correlative relation with the symptoms observed from February 24th until death on February 27th.

2. The aortic endarteritis was in an early stage, but well-marked. It, together with the nodule in the right coronary artery, and that in the spleen, gave rise to the

question of syphilitic disease, but the splenic nodule had not the character of a syphilitic growth; that in the right coronary artery was not decisive, and, in my experience, aortic endoarteritis is a very equivocal change. There was no history of syphilis, and no external indications of it whilst the patient was under my care.

CASE VIII.—*Early stages not under writer's observation. Later, excitement with delusions; still later, exalted notions as to physical and mental powers, incoherence, and mental weakness, great garrulousness, quiet pleased self-satisfaction. Ataxy and paresis, much of the ordinary characters, the gait being considerably impaired throughout the later period. Later still, the patient became more and more demented, and finally passed into a state of amentia, he being also speechless, utterly helpless, and of extremely wet and dirty habits.*

The following were the principal morbid appearances. Some wasting of the brain, and a considerable amount of intra-cranial serum: marked meningeal changes, especially over the frontal lobes, and diminishing considerably from before backwards: atrophy, pallor, and softening of the grey cerebral cortex: diminished consistence of the white cerebral substance universally, as well as of the basal ganglia, of the cerebellum, and of the spinal cord. The adhesions of the meninges to the grey cortex were well-marked. They affected the upper surface of the right frontal lobe more than that of the left, while the lateral (external) surface of the left frontal and parietal lobes was more affected than the corresponding regions on the right side.

C. H. W., Gunner Royal Horse Artillery. Admitted April 27, 1872, then aged twenty-seven years, widower.

History.—This was stated to be the first attack of mental disease, and to have existed since January, 1870, and before coming here the patient had been under treatment at Woolwich, and at Netley; at the latter place from August 11, 1871. The cause assigned for the insanity was "predisposition," the operation of which was supposed to have been "aggravated by domestic trouble."

There was almost no history of this patient's case prior to his admission into Grove Hall, but his delusions, excitement, irrational language, and thickness of utterance were stated to be all of them on the increase at that time. He was also described as beginning to fall about when he attempted locomotion.

State on admission.—Height 5ft. 7in., fairly nourished, equal pupils, skin clear, and thoracic and abdominal viscera healthy, habits clean.

His utterance was imperfect. His tongue was protruded at one's request, but in a jerky manner, and whilst held out was affected with fibrillar tremors and twitchings. His powers of locomotion were affected, and he walked in an unsteady manner.

His expression was usually one of quiet self-satisfaction, of well-being. He was garrulous and passed rapidly from one subject to another, apparently unable to fix his attention for any length of time. He enumerated a long list of persons, and another, of duties, both of them connected with his service in the army. He believed himself to be the possessor of very great physical strength, calling attention to the development of his limbs in proof of his assertion, whilst he was weak in reality. Similar extravagant notions related to his mental powers, and he considered that he had very great intellectual acquirements. Ordinary simple questions he answered correctly, but he became incoherent in continued conversation.

In July, 1872, there was a steadily progressive deterioration in the mental and physical conditions.

By October, 1872, he had become still more completely demented since the date of the preceding note, and was becoming dirty in habits, and helpless, requiring to be washed, dressed, and in every way attended to. The expression and bearing, which formerly denoted a sense of well-being, a quiet smiling self-satisfaction, were now usually indicative of dulness, confusion, and stupidity, in fact he looked bewildered and fatuous. His replies were either utterly irrelevant or merely consisted of "yes," repeated in response to any and every inquiry. When he spoke it was in a slow

and drawing manner, and utterance was also quasi-stammering and muffled. He could manage to stand with the feet kept widely apart, and to take short steps with the feet still kept widely apart, scarcely raised from the floor during progression, and their entire soles planted flatly upon the ground. He would scarcely grasp one's hand, and when he did make the attempt to do so the pressure exerted was very feeble. He was unable to button his own clothing, but was frequently and restlessly fumbling with it. Labial tremors and twitchings were observed, especially during speech, but he would not protrude his tongue.

He was still rather stout, the features, generally, were flabby, heavy, expressionless, and his lips also had lost all their natural flexibility and shapeliness of contour. There were still traces of a fresh complexion, and enlarged cutaneous vessels on the cheeks and nose. Slight left external strabismus was observed, the pupils were about equal, somewhat sluggish, the right pupil becoming the larger in the shade, and assuming an oval and somewhat irregular shape.

By December he was unable to articulate a single word intelligibly; but, without quoting from the various notes made, it may be briefly stated that after the date mentioned in the preceding paragraphs (Oct. 1872) he gradually became more and more demented, helpless, wet and dirty in habits: he also gradually emaciated and finally grew extremely weak, and died on March 26, 1873.

Necropsy.—Thirty-five hours after death.

Head.—The skull was rather thick and dense, the dura-mater healthy. Six fluid ounces of serum drained away from the cranial cavity and brain.

The surface of the convolutions was somewhat flattened. The arachnoid was thickened, opaque, and of a milky hue; these meningeal changes being more marked on the superior and external surfaces of the frontal lobes, less over the parietal, and scarcely at all over the occipital. The meningeal veins were full over the posterior part of the cerebrum. Over nearly the whole superior and external aspects of the brain there was subarachnoid serous infiltration. The membranes were rather friable, but separated from the encephalon with comparative ease. Nevertheless there was considerable adhesion of the meninges to the grey cerebral cortex, and separation of irregular layers of the latter along with the membranes. This affected the superior and lateral surfaces of the frontal and parietal lobes, the summits only of the convolutions being affected. The upper surface of the frontal lobe was more involved in this change on the right side, and on the other hand the external surface of the frontal and parietal lobes had more points of adhesion on the left than on the right hemisphere.

The cortical grey matter of the cerebrum was thin, atrophied, rather pale, universally softened.

The white substance of the brain was softened, as also were the corpora striata and optic thalami. The lateral ventricles contained a considerable amount of serum; the middle commissure was present. The cerebellum was of diminished consistence. The spinal cord appeared to be universally of diminished consistence.

Weight of cerebrum, 40ozs.; of cerebellum, 6ozs.; and of pons Varolii and medulla oblongata, 1oz.

Thorax.—Right lung 24ozs.; left, 20ozs.; their posterior portions were heavy, friable, darkly congested, and oedematous.

Heart.—10ozs. The pericardium over the right ventricle was white and opaque in patches. The walls of the left ventricle were thick, the muscular tissue appearing slightly yellowish. No atheroma. The tricuspid valves were slightly thickened.

Remarks.—1. In its long duration, in the absence of epileptiform or apoplectiform seizures, or of convulsive attacks of any kind, while yet ataxy and paresis are developed in an irregularly progressive but slow manner, and in the post-mortem appearances, as well as in other ways, this case corresponds with those of the second group which have preceded it.

2. It differs, however, from these in the less amount of atrophy of the cerebrum, and in the greater amount of adhesion and cineritious decortication, which in this case were, approximately speaking, somewhat of the more frequent or usual form of distribution.

GROUP III. (See p. 181).

CASE IX.—Loss of memory, dementia, delusions of annoyance and persecution, with depression; quietness at first; later, more marked depression, weeping, and occasional irascibility; attacks of semi-stupor (apoplecticiform) from time to time. Motor signs of general paralysis and somewhat "tabic" gait, slight right hemiplegia. Later, extreme motor failure, and patient bedridden; right-sided convulsions, followed by right hemiplegia; slight conjugated deviation of head and eyes, and left ptosis; modified respiration of ascending and descending rhythm, somewhat excurvated belly, death.

External meningeal changes symmetrically distributed. Adhesion and decortication comparatively very moderate, mainly situated on the left hemisphere, and principally affecting the tips of the frontal lobes, the left temporo-sphenoidal, and the posterior part of the left inferior and left internal surfaces. Marked hyper-vascularity and redness of nearly the whole of the grey cortex of the left hemisphere, and of the greater part of the right frontal lobe, and part of right temporo-sphenoidal. Some induration of the grey cortex of the left frontal lobe, diminishing backwards therefrom. Spinal cord softened, its meninges thickened posteriorly, and the site of flattened yellowish nodules. Slight nodule in cerebral pia-mater and adherent to cortex.

C. E., Private 1st Battalion 11th Regiment. Admitted June 4th, 1875, then aged thirty-two, after fourteen-and-a-half years' service.

History.—This was said to be the first attack of mental disease, to have been insidious in its commencement, and to have first been recognized as fully developed mental disease in February, 1875, but no doubt it had existed for some time previously. The cause assigned for the insanity was "disappointment in love;"—he had been jilted,—but no doubt heredity played an important part in its production, inasmuch as (at least) a maternal aunt had been insane. He had been under treatment at Devonport, and, since May 2nd, 1875, at Netley. He was said to be neither epileptic nor suicidal, to have been of good conduct, latterly of temperate habits, and of indifferent education. His regiment had recently been stationed in Great Britain, but six years of his service had been passed at the Cape of Good Hope and at Hong Kong, and in 1865 he had been invalided home from China for diarrhœa.

When admitted at Netley he was suffering from the physical signs of general paralysis, accompanied by symptoms of steadily advancing fatuity, the exalted delusions and *bien être* being, however, wanting. He decidedly lost ground whilst at Netley, the parietic signs became much more marked, and to the report of his case it was added that "though the evidences of the present attack are said to have been first noticed in February, they must have existed for a considerable time before that." The medical certificates testified to the existence of loss of memory, incoherence, delusions as to annoyances received from other persons, as to the desire of his comrades to murder him, and steadily advancing fatuity, with progressive general paralysis, but as yet without dirty habits.

State on admission.—Height 5 feet 10 inches: weight 157lbs. There was a cicatrix on the glans penis, and a bubo-cicatrix in the left groin. The patient said he had had "the venereal." There were traces of ancient double iritis, the pupillary edges of the irides being very irregular, and frayed, and bound down by whitish strands to the capsule of the crystalline lens. There was a leucomatous patch on the left cornea. Some brownish semi-cicatricial spots existed over the body, of which one or two were in front of the left tibia. The lungs were healthy, the heart-sounds full and loud, the second accentuated, yet the pulse quick. The area of liver-dulness was slightly full, the other viscera healthy.

The tongue was tremulous, and was protruded unsteadily; speech was very shaky and tremulous, and there were twitchings of the lips and face very characteristic of general paralysis. His hands trembled much when he attempted to button his clothing, and the gait was feeble and tottering.

He was very demented and, indeed, showed *great* loss of memory. There were no exalted delusions at that time, and no very decisive delusions of injury, but he said that "the men of his regiment were against him," and began to cry whenever his friends were mentioned to him. He was capable of very little conversation, his ideas being extremely limited.

To summarize the notes :

After admission the pulse remained often rather frequent, but varied from 108 to 72 per minute. The patient continued to be inapt for exercise, prone to sit about listlessly, and the grasping power of the hands was impaired. He also soon became wet and dirty, and took no interest in anything, but wore a dull, anxious, and melancholy expression. His powers of comprehension grew more impaired, and his utterance more embarrassed, and on July 7th he was very feeble and tottering, and was dull, drowsy, and stupid, the pulse was 112, there was difficulty in swallowing, he was wet and dirty, and the extremities were cold. He was ordered to remain in bed for a time, and whilst there he proved to be often noisy and restless at night. Then by the end of the month he was able to leave his bed altogether, the mixture of iron and quassia having been substituted, with advantage to his state, on July 7th, for the potassic iodide and physostigma, which he had taken previously.

He grew stronger and took plenty of exercise for some weeks, but by the end of September he had again become weaker.

In October there was diarrhoea followed by constipation. The right leg seemed to be the weaker of the two, the tongue was protruded with a very slight inclination towards the right side, but there was no decided hemiplegia.

In November he again became bedridden. The gait was extremely unsteady, trembling, jerking, of a somewhat "tabic" character, the feet were planted with extreme tremor and jerky incertitude. The right toes turned out very much; the left did not. The heel was the part of the left foot first brought down in walking, and the ball of the right great toe was, to some extent, dragged along the floor. Left to himself he swayed towards the right side. The grasping power was feeble, but apparently equal in the two hands. On the slightest exposure he shivered much, but independently of that there was extreme tremulousness of the face and lips when in movement, and of the tongue when protruded, and after standing for a few minutes there was general tremor and twitching of the body and limbs. The circulation was weak, the hands and feet somewhat livid, the act of swallowing difficult. He was extremely demented and his replies were only unintelligible mumblings.

November 28th. Slight apoplecticiform symptoms, the patient being drowsy, dull, and fatuous.

During December there was increased weakness of the right limbs.

January 22nd, 1876. This patient had been frequently falling into heavy, drowsy, inattentive conditions, and on this day he was heavy, dull, and stupid in the afternoon, after having been very restless in the morning.

January 23rd. He could not swallow well. The pupils were irregular, small, and sluggish. He resisted passive motion, did nothing he was told to do, took little notice of anything, but was not decidedly somatose. There were tremulousness and subultus of all the limbs. The pulse was 102, regular, soft, and somewhat feeble; soft mucous râles were heard over the bases of the lungs behind.

January 25th. A convulsive seizure occurred which affected the face, mouth, and eyes, especially on the right side, beginning equally in the right eyelid and the right side of the mouth, simultaneously with turning of the head to the right side, but affecting those parts also on the other side. The head and eyes were turned to the right, the eyelids opened and closed rapidly, the face and mouth were spasmodically jerked, the mouth being drawn upward and outward towards the right, and the lips held slightly apart at the right oral angle. The clonic spasms of the eyelids and face were equal in point of time on the two sides, but greater in degree on the right side. The occipito-frontalis was affected about equally on the two sides. The left sternomastoid was firm and contracted, the right was flaccid. The diaphragm and tongue also were spasmodically twitched. (1) The above condition lasted for several minutes. Then (2) the head and eyes turned gradually to the left side, the spasm abated

momentarily, but again increased, and then the right upper extremity became stiff and straight, the hand being on a level with the hip, and being convulsively jerked. Then (3) a stage of more general and more tonic spasm came on, a new convulsion, as it were, beginning, and the head and eyes again turned to the right. The right arm was held out straight and forwards, its fingers being rigid and extended, then severe clonic facial spasm, still most of the right side, and clonic spasm of the right arm and of the diaphragm came on, and then there supervened a return to the condition described in stadium 1. Other changes, and successions of changes, in the convulsions occurred, especially in the right upper limb. They were mainly these. The head and eyes having turned to the right, the right hand was jerked, the thumb was straight and thrust between the fingers, then the right hand opened, the fingers were momentarily extended, somewhat to a position as in the "main en griffe." Then the elbow was flexed at an acute angle; and there was right facial spasm.

This quasi *status epilepticus* lasted 2½ hours. Later in the day other convulsions occurred, the patient remaining in a state of semi-stupor, and incomplete right hemiplegia being observed in the evening.

26th. Convulsions again occurred, mainly right unilateral in distribution, and followed by incomplete right hemiplegia, with slight tendency to conjugated deviation of the head and eyes to the left. Temperature, *right axilla* 99.4°, *left axilla* 99°; pulse 90, breath fetid, gums spongy. The patient was drowsy, dull, stupid, but not unconscious.

27th. Two convulsive attacks occurred on this day, one lasting three-quarters of an hour, and the other one hour and three-quarters. Right hemiplegia continued, and there was now some conjugated deviation of the head and eyes to the left side. The paralysis, well-marked in the right leg and face, was greater in the right upper limb. Dysphagia was present.

29th. Convulsive attacks occurred occasionally. Temperature, *right axilla* 97.8°, *left axilla* 98.3°.

31st. Much the same. Occasional spasmodic jerking was seen about the face and mouth, especially on the right side, and there was marked right hemiplegia with ptosis of the left eyelid. Pulse 114, respiration 28. The loaded colon was relieved by enema.

Feb. 1st. Patient not wholly unconscious; pulse 135, feeble and thready; respiration 30, at times noisy, but variable, and of a peculiar rising and falling rhythm, the early degree, in fact, of "respiration of ascending and descending rhythm." The belly had gradually been becoming excurvated of late. The respiration was mainly thoracic, and crepitation was heard, especially over the lower posterior surfaces of the lungs. The cheeks were flushed. The right eye was occasionally opened very widely and staring, and this recurred during two days. The convulsive attacks recurred, and the patient gradually sank, and expired on Feb. 3rd, 1876.

Principal treatment.—June 9th, 1875, to July 7th, iodide of potassium and phos. stig. ; July 7th until death, ferri perchlor. and quass. On Oct. 19th, potassii iodid. and ammon. carb. were also ordered, and wine towards the close.

Necropsy, 51 hours after death. Body rather thin.

Head.—Calvarium thin; weight of the portion removed, 12½ozs. Dura-mater, ordinary. The arachnoid was slightly opaque and thickened at the base, where, also, there were marked interlobar adhesions. Olfactory bulbs wasted. Patchy thickening and opacity of the meninges between the olfactory nerves and immediately in front of the optic commissure, especially on the right side. On both sides were small, symmetrically placed patches of similar yellowish-white thickening and opacity on the cerebral surface of the arachnoid, viz.: at the outer angle of the orbital surface, on the inner or inferior surface of the temporo-sphenoidal lobe, on the sulcus between the third and the uncinate gyri, and at the outer angle and at the termination of the Sylvian fissures. Meningeal veins full. The pia-mater and arachnoid covering the superior and external surfaces of the cerebrum were thick, tough, and faintly opaque, these changes being most marked over the frontal and parietal lobes. They were also very fairly marked over the temporo-sphenoidal lobe, and over the internal surfaces of the hemispheres, except the posterior half-inch. The membranes

covering the tip of the occipital lobe escaped this change. The membranes as a rule stripped fairly, leaving shreds in the sulci, and were rather hypervascular. The "arachnoidal villi" were well developed. The pia-mater was slightly cedematous in the fronto-parietal region.

There were slight meningeal adhesions to the cortex at the tip of the frontal lobe on both sides, more marked, but within more narrow limits, on the left first frontal than on the right, and very slight on the orbital surface. Adhesion was seen also on all the convolutions of the external surface of the left temporo-sphenoidal lobe, but only very slightly on the first convolution, and that in front: of the right temporo-sphenoidal lobe it was only seen on the second gyrus: also on the upper part of the posterior half of the internal surface of the left hemisphere: very slightly on that of the right, and also was it observed at the upper end of the right ascending parietal gyrus. There were a few scattered adhesions on the inferior surface of the right hemisphere, and more decided adhesions scattered over the posterior third of the inferior surface of the left hemisphere.

Speaking generally, numerous vessels were visible to the naked eye in the grey cortical substance giving it a dull pinkish hue. It was of fair thickness, the convolutions were fairly developed, and the stratification fairly obvious.

In the *right* hemisphere the grey matter of the superior surface of the frontal lobe was generally reddish, but that of the ascending frontal gyrus and of the posterior part of the third right frontal was of a dull yellowish fawn colour, and its strata were very obvious. Behind the frontal lobe the colour was nearly natural or only slightly reddish. The grey cortical matter at the base of the brain was of natural hue, except the anterior inch-and-a-half of the temporo-sphenoidal lobe which was red.

In the *left* hemisphere the whole of the grey cortex of the superior and external surfaces was of a deep dull pinkish tinge, with the exception of the upper ends of the two ascending convolutions. The grey cortex was of firmer consistence in the left than in the right frontal lobe. This undue firmness of the left grey cortex diminished from before backwards to a natural consistence at the occiput. The colour of the cortex was also of a darker reddish hue here than in the right hemisphere.

The white substance of the brain was moderately vascular, the vascularity being greater in the left hemisphere; its consistence was lessened—a point in which it differed from the grey cortical substance.

The grey cortex of the lower tiers of convolutions of the posterior half of the right hemisphere, and that of the posterior half of its inferior surface, was pale. In the corresponding parts of the left hemisphere the grey matter was, for the most part, dark red. On the inferior surface of the left hemisphere the grey cortex was firmer than natural in front, slightly so at the posterior part, and slightly softer than natural in the intermediate temporo-sphenoidal region.

The fornix was soft, the ependyma ventriculorum was firm. At the edge of the sulcus between the first and second frontal gyri, and on the second, $1\frac{1}{4}$ inch from the tip of the frontal, was a flattened firm yellowish-white nodule embedded in the pia-mater, and at that point the pia-mater, arachnoid, and grey cortex were all closely adherent to each other.

Weights.—Right hemisphere, 20½ozs.; left ditto, 20½ozs.; cerebellum, 5ozs.; pons and medulla oblongata, ¾oz. Fluid from cranial cavity, 2½ fl. ozs.

The spinal cord was softened generally—certainly in its upper portions. Over the greater part of the length of its posterior surface the arachnoid, from posterior roots to posterior roots of the series of spinal nerves, seemed to be greatly thickened, opaque, increased by adventitious layers of a gelatinous, faintly yellowish-grey, colour, and adherent to the pia-mater of the cord. But about 4 inches, and, again, about 6 inches, from the lower end of the medulla oblongata, the meninges were more elevated, were highly vascular, and bulged behind, and in them, at these points, were embedded two flattened, firm, yellowish-white masses. These nodules stripped off along with the arachnoid, and were separable from the pia-mater.

THORAX.—Heart. Weight, 10½ozs. Slight aortic atheroma. Valves, healthy. Muscular substance of heart slightly softened.

Left lung, 25½ oss.; some old pleuritic adhesion. The posterior part of the lower lobe was inflamed. The apex was slightly puckered, and its summit was nearly consolidated by firm very small tubercles, some whitish, some greyish. *Right lung*, 26½ oss.; much the same as the left, except that it contained no tubercle.

Spleen, 6½ oss. A cartilaginous thickened patch on the capsule. *Kidneys* congested, left 8ozs., right 5½ozs. *Liver*, 56ozs. A little turbid serum was found in the right side of the peritoneal cavity. The small intestine was gathered into the pelvic cavity, and in parts of it there was patchy congestion.

Microscopical examination:—

(A.) The grey cortex of the posterior part of the *left* third frontal gyrus showed slight granular degeneration of some of the nerve-cells. There was hyperplasia of the nuclei of the walls of the vessels, and the parietes of some of the small vessels were thickened, and some of these had also dark grey deposits in their walls. The neuroglia had also undergone overgrowth and increase. Some "colloid" bodies were observed.

(B.) Now in the corresponding part of the *right* third frontal gyrus the grey cortex was much paler, and was softer than in the left; that is, it was of natural consistence, and not slightly indurated as on the left side. In some respects the microscopical appearances were the same; thus, vessels were found with increase of nuclei on their walls, and here and there molecular deposits therein.

The microscopical differences between the two were:—

α. Partly in the nerve-cells. Here (*right* third frontal gyrus) they were scarcely granular, or were much less so than in the corresponding part on the left side. A large proportion of small pyramidal cells were seen, but only very few of the somewhat square-shaped nerve-cells.

β. Partly in the smaller number of neuroglial nuclei, whatever their shape, on this right side, where also the connective tissue lacework was less observable.

γ. Partly in the fact that no colloid bodies were seen here, while they were present on the left side.

(C.) In the portions of the spinal cord examined were compound granule masses and colloid bodies, and some of the nerve-cells were cloudy and more or less granular.

(D.) The nodules from the spinal meninges were vascular and contained much connective tissue. A few of the vessels were thickened. There were pale, rather ill-defined, small, oval, free nuclei or corpuscles, and also some scattered nodules, and small granular heaps.

Remarks.—1. As to the question of syphilis, which of course arose in this case.

In support of the view that it was present there were during life only, (I.) the patient's assertion that he had had "venereal,"—(II.) the penile and inguinal scars, and (III.) the traces of old double iritis. Of these the first was vague and unreliable owing to the mental state of the patient, the second bespoke the non-infecting venereal sore rather than syphilis, and the third was equivocal.

Then after death there were the firm yellowish-white flattened nodules in the spinal and cerebral meninges. These were perhaps syphilitic, though not unlike yellow tubercle.

The patchy and marked opacity of the cerebral arachnoid favoured the hypothesis of a syphilitic origin.

2. The chronic spinal meningitis and other changes in the spinal meninges, and softening and other morbid conditions of the spinal cord, were probably in relation with the early and marked feebleness of the limbs—the grave paresis.

3. Of further interest was the question whether the diseased condition of the posterior part of the spinal meninges and the nodules in them were the cause of the markedly "tabic" or "ataxic" gait by their pressure-effects on the posterior columns of the cord, or whether this form of gait must in this case be assigned to changes seated in the posterior columns themselves, or in the ascending (parietal) convolutions of the brain.

4. Another problem presented itself in the inquiry whether any, and if so what, relation subsisted between the less usual character of the mental symptoms,

—namely the complete absence of exaltation and of exalted delusions, and the presence of much dementia, with delusions of fear and persecution, on the one hand;—and, on the other hand, either the predominance of morbid change in the left hemisphere as evidenced by (a) the adhesions and by (b) the morbid redness, hypervascularity and induration; or, again, the unusual involvement of the posterior part of the left hemisphere in the adhesive change.

Of these changes the adhesion and decortication principally affected therein the tip of the frontal, the external surface of the temporo-sphenoidal lobe, the posterior half, or less, of the inferior surface, and the posterior half of the internal surface. The cortical discoloration was very general in the left hemisphere, and the slight induration of the grey cortex gradually diminished from before backwards.

The third kind of morbid alteration referred to—that of the posterior part of the inferior and internal surfaces—is too rare to speculate about.

Certainly the marked predominance of the changes in the left hemisphere is an important point in the case.

5. Still another question was the relation (if any) between the lesion described as predominant in the grey cortex of the left hemisphere, and the convulsions, which were mainly unilateral and dextral. Or, again, the relation (if any) to the same convulsive movements, of the nodule in the meninges adherent to the left second frontal gyrus. These are questions of special interest to determine by a consideration of the beginning and march of the spasm, and of the eye and arm movements; and others.

The connection, I take it, was between the convulsions and the former and more diffused morbid change; rather than between the convulsions and the irritation of the cortex, and the subsequent changes induced locally, by the nodule specified. For the latter escaped what (in monkeys) is called the motor region, and though of course the effects of its irritation might have spread to the neighbouring parts within the motor zone, yet the beginning and march of the spasm, the order in which the several parts were affected thereby, did not correspond to what would have been anticipated, if one viewed the nodules as the focus of irritation causing those functional changes in the grey substance which led to convulsions, and expected to see the latter follow the course of those produced by stimulation of the corresponding region in monkeys. ("The Functions of the Brain," Ferrier, p. 143.)

6. The slight but varying right hemiplegia, which lasted for many weeks, was apparently dependent upon the somewhat diffused, yet partially circumscribed, lesions in the grey cortex of the left hemisphere.

7. The highly-marked right hemiplegia of the last few days of life was evidently of epileptic origin, supervening on convulsions which were mainly dextral in their range.

8. The microscopical, like the naked eye, differences between the two cerebral hemispheres showed that the left was the more diseased.

CASE X.—Mental disease insidious in onset, characterized by eccentricity of conduct, and then marked mental disorder, culminating in a resolute suicidal attempt. Subsequently, melancholic delusions, incoherence, loss of memory, depression, anxiety, and alarm, and hallucinations of sight. Later on, the patient was garrulous, querulous, worried, dejected and lachrymose; but now and then displayed some largeness of idea, and was also restless at night, destructive, and even violent. Later on, he was confused and childish.

Motor signs of general paralysis of the first and second stages; much motor restlessness; and finally apoplectic cerebral congestion with hemiplegia of the common form, ending in death after four days, the accompanying conjugated deviation of the head and eyes being only slight and temporary.

Intense meningeal engorgement. Meningeal (chronic) changes not well-marked, but especially observed over the mid-vertex. Encephalon engorged by mode of

death, especially on the right side. The grey cortical substance of the cerebrum was thinner in the left frontal region than in the right, but was of about the same depth on the two sides in the posterior part of the parietal and in the occipital lobes. Grey matter rather firm, especially in the upper layers on the right side, and especially in the frontal lobe on the left. Left hemisphere less in weight. Adhesion comparatively slight, and affecting first and second frontal gyri only.

R. T., Sergeant 48th Regiment. Admitted Aug. 13th, 1875, then aged 37 years, service, 17½ years, married.

History.—This was said to be the first attack of mental disease and to have existed in a marked form from the beginning of July, 1875, but its origin had been insidious and dated some time further back. It was supposed that the mental derangement was brought on by disease contracted at military stations in India. He was said to be suicidal.

Invalided home from India and transferred from the surgical division to the lunatic wards at Netley on June 30th owing to certain peculiarities of conduct, he slowly and steadily deteriorated both in mental and in physical condition. As he seemed to be harmless it was in contemplation to hand him over to the care of his friends, when, on the night of August the 7th, he made a determined attempt at self-destruction by hanging with sheets. He was therefore transferred to Grove Hall, certified as being the subject of hallucinations of sight at night, and of delusions as to every one being hostile to him, and as to poison being placed in his food. It was also certified that he was incoherent, wandering from subject to subject in conversation, that he could not settle to any work, but showed a propensity to wander about, was suspicious of harm from all those about him, had on the same night attempted suicide and violently assaulted an orderly, and that he showed twitching of the muscles of the lips and tongue accompanied by thickness of speech, indicating an early stage of general paralysis of the insane.

Mute on admission.—Height, 6ft. lin.; weight, 156lbs. The head was of narrow dolichocephalic shape, and the sagittal suture was high and ridged; this helped to give a carinate and pentagonal form to the skull. Pupils nearly normal, face slightly pale and thin. The speech was thick and tremulous, and very characteristic of general paralysis; the patient was voluble and spoke rapidly, but the peculiarity of speech, and the tremors of the face, lips, and tongue were greatly increased after active exercise. The pulse was 120, quick, compressible; the heart's action irritable, its second sound clear, but not accentuated. The viscera appeared to be normal. Sudamina were seen on the chest.

The right side of the mouth acted slightly better than the left, and the hearing was defective in the left ear, from which the patient said there had been a discharge.

There was a history of a venereal sore on the penis about fifteen years before, of which no decided trace remained, but in the left groin were two large bubonic cicatrices. There were no indications of syphilis. He suffered from no headache or pain, nor from numbness of any part. There was no decided local palsy. The veins of the legs were varicose.

He grasped very powerfully with the hands; the gait was strong and he walked with confidence, but there was some unsteadiness in turning round, and he marched rather heavily.

He declared that "every one was against him" and put poison into his food. He did not feel safe, and begged for protection against his enemies. He was restless, excited, garrulous, engaged frequently in rambling conversation, and gave long disconnected accounts about himself consisting of delusions as to his treatment, both in India and since, quorulously bringing charges against all who had had the care of him in any way. His expression was anxious, agitated, and dejected.

Aug. 23, 1875. The patient had been restless since his admission, and full of complaints about his detention. Two days before this date he had been very restless, excited and loquacious; he smashed a window in his dormitory, and was sent to sleep in a padded room. He had taken chloral hydrate and bromide of potassium

and had become calmer. Pulse 114, head somewhat warm, face pale, speech markedly and characteristically affected. He was suffering much mental malaise, anxiety, and worry, and yet contemporaneously with these were sometimes statements which evinced a feeling of well-being and rather large ideas.

During September my notes mention occasional restlessness, excitement, and self-stripping of clothes. At night the restlessness was greater, and he was then subject to hallucinations of sight. In conversation he was childish, confused, and now and then would break into tears. He was taking digitalis, and the pulse varied from 76 to 90 on different days.

Early in October the pulse was tense, full and unduly frequent, the patient was less restless and excited, but worried himself a good deal, and was disquieted about various subjects.

Oct. 21st. The patient had been violent the day before, and heavy and stupid at night. He was now lying in bed, in a state of stupor, but could be roused slightly, and he made a slight attempt to put out the tongue; the respiration showed a slight tendency to stertor. The pupils were sluggish, of medium size, the right pupil being very slightly the larger, the left having been slightly the larger as a rule. The left arm appeared to be more helpless, non-resistant, and flaccid than the right, and the left lower limb, also, was slightly palsied. The face was diffusely injected, so was the conjunctiva. The temperature in the left axilla was 103.2° , in the right 103.1° .

Ordered saline aperients and enemata, croton oil, bromide of potassium, veratrum viride, and ice to the head.

At midnight the pulse was 84; the pupils were equal, rather small, and quite immobile; respiration was 18, quiet, regular, and there was slight puffing of the cheeks in expiration. The left arm was the more paralyzed, there was no palsy of the face, the left lower limb was slightly the less resistant to passive motion. The stupor had continued during the evening; ice had been kept to the head, and the temples and nostrils had been leeches. For a time the left arm was rigidly bent at a right angle at the elbow-joint, the hand being pronated, the thumb and forefinger being kept stiff and straight, and the outer three fingers being flexed. Continue the mixture and ice, and apply sinapisms to the feet.

Oct. 22nd, 9 A.M. The urine was retained and five pints were drawn off by the catheter. 1 P.M. Pulse 86, rather full, hard, and long. Respiration 18, the cheeks puffed a little in expiration and saliva was blown upon the lips. The bowels had been relieved seven or eight times during the past twenty-eight hours, the face was still red and suffused, the body warm, but the feet, especially the left, slightly cold, the left arm limp and palsied, and the left leg also, but in less degree. The mouth was somewhat drawn to the right. Slight temporary conjugated deviation of the head and eyes was observed. During the night, and on this day, there had been occasional convulsive jerking and tremors of the right arm. Now and then the patient would protrude the tongue momentarily at request, or in reply to a question would mutter the words "all right" or "no." The temperature in the left axilla was 103.2° , and in the right axilla 102.9° . Treatment continued. Oct. 23rd. Much the same. Pulse 96, smaller. Respiration 26, slightly stertorous. He could still be partially roused as on the 22nd, but the tongue was not protruded. Temperature, left axilla, 101.8° ; right, 101.7° . There was left hemiplegia, the right arm resisted passive motion. There were signs of pulmonary congestion and oedema.

Oct. 24th. The urine was acid, and contained excess of urates and of phosphates. There was much hiccough. Pulse, 78, softer and smaller than before; respiration 28 and inspiration more noisy; temperature, right axilla, 100.3° ; left 100.2° . Much flatus was passed downwards. The pulmonary signs were increased. At 6 P.M. was more comatose, swallowed very badly, had great gaseous distension of abdomen (Terebinth). Temperature, left axilla, 100.2° .

Oct. 25th. Patient worse, more comatose, sweating freely, face and ears livid and purplish; pulse 130, full and bounding; respiration 49, loud and laboured; severe hiccough, mucous rattling in throat, pupils sluggish, rather small. Temperature, right axilla, 101.3° ; left, 102.5° ; general passive congestion of the veins; abdomen

highly tympanitic and swollen. The patient became more comatose, and died at 5 P.M. on Oct. 25th, 1875.

Necropsy, 21 hours after death. Calvarium congested. Sinuses of dura-mater and meningeal veins deeply gorged with dark fluid blood and clot.

Right hemisphere of cerebrum.—Weight 23½ ozs. The convolutions were fairly plump. The membranes stripped with some difficulty, left shreds in the sulci, were slightly oedematous over a limited part of the parietal and frontal lobes, were slightly both thickened and tough, presented the faintest trace of opacity over the mid-region of the convexity, and were not adherent except at a few small points at the upper part of the tips of the first and second frontal gyri, especially of the latter. The grey matter was of a deep pinkish hue, particularly in the frontal region, and showed numerous visible vessels. In the upper parietal convolutions the colour was much paler, especially in the deeper layers. The colour was again deepened in the occipital region. The grey cortex was of ordinary depth, and was of increased consistence throughout this hemisphere, and particularly so in its superficial layers, and in the gyri at the vertex, and in the frontal lobe. The strata were indistinct. The grey cortex of the inferior surface was firmer than usual in the anterior region, but less so in the middle and posterior regions.

The white matter was firm, of a mottled violet and lilac hue, and hyperæmic, especially near the base of the brain. There was an old adhesion between the intra-ventricular part of the corpus striatum and the wall of the ventricle. The corpus striatum and optic thalamus were plump, firm, hyperæmic, and presented numerous puncta cruenta and dilated vessels. The lateral ventricles were nearly empty.

Left hemisphere of cerebrum.—Weight 22½ ozs. The membranes here were as on the right side. Adhesion was almost limited to the upper part of the tip of the first frontal convolution. The convolutions were depressed below the level of the adjoining ones over an area of about half an inch square, immediately before and behind the external parieto-occipital fissure. The grey cortical substance was paler on this than on the right side, its layers were not nearly so thick as that of the right, in the frontal lobe, and of about the same depth in the posterior part of the parietal and in the occipital lobe. The depth of the grey matter gradually diminished as one passed forward from the parietal lobe. The grey matter was unusually firm in all the upper regions of the cerebrum, especially in the frontal lobe where the colour was ordinary with a faint slaty tint in the inner layers. Few vessels were visible to the naked eye in the grey matter, on this side, and there was a slight pinkish hue only in the posterior of the parietal and in the occipital convolutions. Elsewhere the colour was about ordinary.

The white matter was much the same as on the right side, and the radicles of the *vena Galeni* were equally engorged on the two sides.

The corpus striatum and optic thalamus were much paler than on the right side. Still they were firm and hyperæmic.

The grey cortical matter at the base of the cerebrum was thin, firm, and of ordinary hue in the orbital region, but thicker, softer, and more vascular over the base of the temporo-sphenoidal and occipital lobes and at the back part of the internal (median) surface.

The pons Varolii and medulla oblongata were slightly congested. Weight 1oz. The medulla oblongata was very firm, and the tissues beneath the floor of the fourth ventricle were pinkish.

Cerebellum, 6ozs., its white matter rather firmer than usual.

THORAX.—*Heart* 11½ ozs., its muscle healthy; 1oz. pericardial fluid. The left ventricle was contracted. The mitral valves were thickened, and the aortic slightly so. Immediately above the aortic valves there was an elevation of the internal surface of the aorta containing a calcareous mass; and another of fibroid appearance.

Right lung.—Weight 39½ ozs. Hypostatic congestion and pneumonia.

Left lung.—Weight 37½ ozs. Much the same. In the lower lobe part of the pulmonary tissue was breaking down as if about to form a cavity, with irregular walls, dark grumous contents, and a gangrenous tendency. There was also slight pleurisy at the lateral surface.

Abdomen.—The *intestines*, distended with gas, thrust the diaphragm upwards.

Kidneys, healthy. Right, 6 ozs. ; left, 6½ ozs.

Spleen, 7½ ozs. A few old filamentous adhesions, and patches of slaty-grey thickening of the capsule.

Liver, 84½ ozs. Old long filamentous and membranous adhesions of the upper surface of the liver to the diaphragm and abdominal walls. At the point of adhesion of one of the largest bands to the liver was a cicatrix with thickening and opacity of the capsule there, the cicatricial tissue passing to the depth of ¼ inch into the hepatic parenchyma.

Microscopical examination of hardened and stained sections.

Tip of *right* second frontal convolution :—The nerve-cells were abundant, were slightly granular, and there was a want of clearness in their contours. The nuclei of the neuroglia were numerous. Many little vessels contained clot and were gorged with blood corpuscles, and one showed an appearance of rupture. In other parts of this gyrus were slight fusiform vascular dilatations.

Tip of *left* second frontal convolution :—The want of clearness about the contours of the nerve-cells was more marked on this side. The other changes were much the same.

Right ascending parietal convolution. Appearances much the same as the preceding. Some of the nerve-cells were granular and their nuclei obscured. Deposits were observed on the walls of some of the vessels, and at one point a minute vessel seemed to have given way, and permitted of slight extravasation into the nervous substance.

At the tip of the occipital lobe nothing special was found.

The pia-mater over the tip of the left frontal lobe showed vessels with hypertrophied walls, some tortuous, and some bulged here and there with blood corpuscles.

Remarks.—1. In this is an example of general paralysis terminating after a short course as if by an accidental complication, for apoplectic cerebral congestion, a common, and often a recurring, phenomenon in general paralysis, assumed a fatal intensity in this case.

2. On the one part may be seen an intense *symmetrical* meningeal congestion, and yet on the other marked hyperæmia and a normal depth of the cortical grey substance of the right hemisphere, and hyperæmia also of the right opto-striate body, contrasting with the paler corresponding parts in the left hemisphere, and with the thinner, atrophic, but equally firm, grey cortical substance of the anterior part of the left hemisphere. To this hyperæmic, and probably, in part, passive engorgement of the right hemisphere may be assigned the production of the left hemiplegia during the fatal attack.

3. There were traces of old perihepatitis and perisplenitis. These had, perhaps, been connected with tropical diseases contracted in India, and all the more so inasmuch as no traces of syphilis were observed, and no decided history of it forthcoming.

4. The great gaseous distension of the bowels hurried on the fatal termination by way of respiratory limitation and circulatory obstruction. And the invasion of the chest by the liver, thrust upward with the diaphragm, led to the question of the diagnosis between this condition on the one hand and pleuritic effusion on the other, with either collapse of the base of the lung or hypostatic pneumonia.

5. The morbid process attacking the left in advance of the right hemisphere had already occasioned some atrophy, and had probably lessened the vascular dilatability therein.

CASE XI.—*Considerable mental impairment, defective memory. Insomnia, and fidgety excitement. Listlessness and wandering habits. Transient, or doubtful, extravagant notions, the patient, later on, being usually dull and stupid in appearance, but sometimes nervous and alarmed, quiet by day, but at night shouting or hiding away in confusion, alarm and terror. Self-stripping, destructiveness,*

degraded habits. Finally, extreme dementia, with helplessness, and obtuse sensibility. The motor signs of general paralysis were of the ordinary character, but to these was added more or less incomplete slight hemiplegia, which grew worse paroxysmally and perhaps never entirely disappeared. Latterly, the resistance to all manipulation was a very marked feature. General tremulousness on exertion was observed nearly throughout.

Greater adhesion, decortication, and atrophy of the left than of the right hemisphere, the left lateral ventricle being also the larger, and the left corpus striatum the smaller. The adhesion was extremely wide-spread and highly marked, existing to a considerable extent in the anfractuositities as well as on the summits of the gyri, and being unusually well-marked over the base of the brain. The grey cortical substance rather pale, but reddish and vascular in parts, atrophied, of ordinary consistence. White cerebral substance pale, and perhaps slightly firmer than natural. Meninges pale, thickened, and in parts œdematous, and considerably changed over the base of the brain.

J. J., Private 2nd battalion Coldstream Guards. Admitted July 10, 1876, then aged 27, 9½ years' service; "home" stations only. Single.

History.—This attack of mental disease was said to be the first, to have come on insidiously, to have existed during all that portion of 1876 that had then passed away, and to have been treated, before coming to Grove Hall, at the Guards' Hospital in London, and then at Netley since May 8. The cause was stated to be unknown. The patient was said to be neither suicidal nor epileptic, and to have been of good character and temperate habits. No family history was procurable. There was no record in his medical history sheet of epilepsy, injury to the head, or sunstroke, but he was said to have suffered from constitutional syphilis, and there was a record of his admission to hospital during six days for "paralysis of the right side," the cause of this palsy not being stated.

Whilst the patient was at Netley there was impaired power of the right side, and the grasping power was sensibly diminished; there was a slight halt in the gait. He was said to have exhibited, also, the signs and symptoms, physical and mental, of general paralysis, "with the exception of the delusions of grandeur." It was also added in the report, that whilst there "treatment had consisted in the employment of chloral hydrate and tincture of calabar bean, but no good effects had been obtained."

The certificates upon which he was sent to Grove Hall testify to:—

1. Great confusion of thought, very defective memory, insomnia, a fidgety manner, occasional excitement, propensity to wander about in a listless objectless way, imperfect articulation, tremor of the muscles of the tongue and lips, incomplete paralysis of the right side, and advancing general paralysis of the insane.

2. Great hesitation in speech and nervousness, trembling of hands. The statements made by the patient that he had found large quantities of diamonds in the yard of the hospital, and that a woman came at night and stole them; also that a woman was in the habit of coming at night and making his bed.

State on admission.—Physical conditions.—Height 5ft. 11in., weight 168lbs. The tongue was protruded slightly to the left side, but the right side of the mouth was the one less fully drawn up at request, while on the left side the lips fell slightly together. The grasping power of the right was less than that of the left hand, but of both was fair; the right leg seemed to grow fatigued the more quickly of the two, and then the gait became limping in that limb. The pupils were rather small and were susceptible. When protruded the tongue showed a slight general tremor, speech was hesitating, quasi-stammering and jumbled, shaky and slow. There was much labial, and with it some facial, tremor, especially just before and during speech, the writing was shaky, any muscular action was accompanied by marked tremor of the muscles engaged therein. He was restless and shaky, and tremor was easily induced. The features were somewhat broad and of deficient expression.

The heart's sounds were full and loud, the second was accentuated and heard widely, the pulse was of ordinary characters. There were no signs of syphilis, he denied having ever had it, and also denied having had any headache or cranial pains.

His mental operations were slow and impaired, though his memory for leading events, and on simple matters was still fair, but he forgot many things he should have recollected, as for example the name of the colonel of his regiment. He exhibited no pleasant joyous excitement, no exaltation, and no grandiose delirium. With reference to what was mentioned in the certificates about diamonds, as noted above, he now said that a woman stole some valueless pebbles from him. His ideas were confused, he said he had been "on guard" the preceding day, and that he "was tormented by a woman at night." Sometimes he looked startled, nervous, confused,—at others, dull, stupid, and void of the expression of any feeling.

Such was his usual condition during the day, but *at night* he would often get up in terror, declaring that "some persons were after him," or shouting "murder," and wildly endeavouring to get away, and on one occasion he smashed the glass of his dormitory window whilst in this state of terror. Sometimes, however, he would hide his head under the pillow and huddle up underneath his bedding, apparently under the influence of morbid fear.

July 24th, 1876. The patient was heavy, drowsy, had a dull and vacant look, and the face was flushed. This passed off.

August 3rd. At times he was restless, confused, meddlesome; at others agitated, and in terror. Occasionally he was busy at night, pulling his bedding about, and on one such occasion was found bathed in profuse hot perspiration. His movements were very tremulous, and he fumbled much, especially when he attempted to handle anything, and the tremulousness was also marked during speech. The dementia and failure of memory, and the cardiac sounds were still much the same as when he was admitted.

August 8th. He still was occasionally confused, restless, meddlesome by day, and apt to tear his clothing. The trace of former hemiplegia was now only recognizable in the face, and was only very faintly marked there. He was so restless, wakeful and destructive at night that hypnotics were ordered.

August 16th. Had again been restless and "bed-making" at night, and was destructive to clothing. He was confused, flushed, and heavy.

September 25th. From time to time he partially lost power in the right limbs, and for some little time now had scarcely ever entirely regained it.

November 1st. He was very restless, seizing hold of and pulling at everything about him. Was still allowed to sit up, but in moving he stumbled in a helpless manner, and was helpless generally. The face was often flushed, and on these occasions he was restless, obstinate, and the mental functions were more than usually obscured. So stupid and fatuous was he, as well as restless, that he was very difficult to manage, so much did he resist manipulation. Immediately after this note, and on November 3rd, he became bedridden, and remained so until his death. Early in November vesical catheterism was required, and slight bedsores formed with extreme readiness. Cutaneous sensibility had become obtuse.

January 1st, 1877. He now was utterly fatuous and rarely spoke, but ground his teeth together with great constancy and vigour. There was slight right hemiplegia, which paroxysmally became worse, and there was always marked tremulousness of the muscles when movement was attempted.

By January 14th there was hypostatic pneumonia, especially of the right lung, and a large abscess had formed also in the left thigh, connected with the ulcerated surface over the left hip. This was opened. R. Quinine and Sp. Vini Gallici.

Emaciation became extremely advanced, the pulse became very weak and rapid, only a little fluid could be swallowed, and the patient, gradually becoming more exhausted, died on January 19th, 1877.

When first admitted this patient was placed on potassic bromide and physostigma; later, on K.I. and Fe.; finally on Ferri perchlor.

Necropsy.—Sixty-eight hours after death. Body emaciated.

Head.—Calvarium thin, the portion of it removed weighed 15ozs. The dura-mater was in the ordinary condition, but pale. General slight thickening and opacity of the arachnoid existed over the base of the brain, where also the membranes were pale, and interlobar adhesions were well-marked. The arachnoidal villi were of

very full size. There was a milky hue of the arachnoid over the whole of the superior and lateral surfaces of the cerebrum, and the combined pia-mater and arachnoid were thick, tough, anæmic, and the former partially oedematous, the rather wide and rounded sulci being filled with serous fluid.

The membranes were closely adherent to the surface of the cerebrum, and a considerable quantity of the cortical grey matter stripped off with them when they were removed, not only from the summits of the gyri, but partially from the sides of the anfractuositics between the gyri as well. This adhesion and decortication occurred over every surface of the left hemisphere of the cerebrum, upon its superior, inferior, lateral, and internal aspects, being especially marked over the parietal lobe and part of the frontal lobe, and almost as much over the tip of the frontal, somewhat less over the occipital, and still less over the temporo-sphenoidal lobe. On the inferior surface of this left temporo-sphenoidal lobe there was more adhesion than on the lateral (external) surface. The same condition and the same general distribution of it were found in the right hemisphere of the cerebrum, but less marked and less extensive than in the left. The grey cortical substance was of a slightly reddish tinge in parts, but as a rule was rather pale; its consistence was ordinary, but it was rather thin.

The white cerebral substance, generally, was rather pale, and perhaps unduly firm.

The lateral ventricles were much dilated, particularly the left one, and contained more than a fluid ounce of serosity. The choroid plexuses were pale and shrunken; the grey commissure was present; the veins overlying the walls of the lateral ventricles contained but little blood. The left corpus striatum was slightly shrunken, and all the basal ganglia were pale on section, although the vessels of the corpora striata contained stringy clots.

The cerebellum was rather softened, and its grey matter pale. Weight of right cerebral hemisphere, 21½ ozs.; of left, 20½ ozs.; of cerebellum 5½ ozs.; and of pons Varolii and medulla oblongata, 1 oz. 6¾ fl. ozs. of serosity escaped.

Thorax.—*Heart*, 9½ ozs. The blood was nearly all fluid. On the inner surface of both coronary arteries, especially of the left, were raised, opaque, whitish patches. The inner surface of the arch of the aorta had an irregular modulated appearance, produced mainly by greyish and somewhat translucent tissue containing yellowish and opaque patches and spots, and, in parts, by superficial yellowish patches, easily detached from the subjacent tissues. The muscular substance of the heart was rather too friable.

Left lung.—Weight 31½ ozs., slight apex adhesions. Scattered throughout the upper lobe were a few opaque, white, firm, and in parts cheesy nodules; the posterior portion was of a deep purple hue, firm, sank in water, and had numerous nodules of pneumonia dispersed throughout it.

Right lung.—56 ozs., much congested. More advanced cheesy and pneumonia changes, passing at parts into excavation.

Abdomen.—*Spleen*, 15 ozs. of deep purplish chocolate hue. The *kidneys* each weighed 6¾ ozs., and appeared to be healthy. *Liver*, 53 ozs. A starlike thickening of the capsule was observed at one point, but not extending into the gland.

Remarks.—1. From time to time right hemiplegia, incomplete in degree, and, oftentimes, partial in extent was observed. Now and then it almost absolutely disappeared, and anon it became paroxysmally worse. As far as revealed by the necropsy its occurrence might be explained by several conditions, any one or all of which might be assigned as the cause, according as we adopt this or that view of cerebral pathology.

a. By the great morbid surface change of the left cerebral hemisphere.

b. By the greater atrophy of the left cerebral hemisphere.

c. By the slight relative atrophy of the left corpus striatum.

d. By the reflex effect of irritation by some portion of the organic disease observed.

2. The co-existence of aortic endoarteritis (nodulation) with the statement that the patient had had constitutional syphilis could not be lightly passed by. Yet there were no indications of syphilis during life whilst the patient was under my

care, nor any unequivocal indication of it after death, unless the aortic endoarteritis be assigned to that cause. True it is, that the meningeal adhesions were unusually well-marked, extending, indeed, into the anfractuositities, and therefore indicative of an active adhesive and probably inflammatory process at some period. But, whatever its cause, the change was of a kind common in general paralysis, and I could find no absolute proof of an intra-cranial syphilitic lesion. True it is also, that the uniformity of the hepatic capsule was relieved by the star-like, thickening in its upper surface; but, again, no trace of syphilitic gumma or inflammation was to be seen, there was no cicatrix extending into the hepatic parenchyma, no other evidence of perihepatitis, whether alcoholic or of another kind, and milky circumscribed thickenings of this description may be due to some compression, and consequent friction, during locomotion, owing to unaccustomed conditions of effort at some period of life, as, for example, when soldiers are drilled invested with accoutrements.

CASE XII.—*Insidious origin, long duration. At first, eccentric conduct, gusty fits of passion and destructiveness, mistakes in, and unfitness for, performance of duty. Impaired perceptive, mnemonic, and reasoning powers. Incoherence, childishness, dementia, docility, obedience, the countenance generally expressive of placidity and pleasure. At first a varying state of dysphasia,—later the speech much impaired. Severe early headache, and, later, recurring headaches, after which the patient was for a time unusually dazed and confused. Extensive incomplete anæsthesia. Tremor c̄actus of hands and arms. Seizure of unilateral convulsions and of local spasm, long continued, changing from part to part and especially affecting the right side. Right hemiplegia variable, and recurring, but too persistent to be purely epileptic. Helplessness, rigid flexed contraction of limbs, especially of the right. Finally, vomiting, low temperature, and slow and feeble pulse.*

Meningeal changes, symmetrical in external appearance. General paleness of brain. Left grey cortex paler than right, and especially so in the frontal lobe, and of this lobe especially in the third frontal gyrus. White substance, pale and very slightly indurated; this more obvious in left frontal lobe. Left hemisphere 1½ ozs. less than right. Adhesion and decortication, especially of prominences of gyri of superior and lateral surfaces of cerebrum, and principally in the parietal lobe, less in the temporo-sphenoidal and frontal lobes, slight in the anterior half of the frontal, invading the occipital, affecting the posterior half of right slightly more than left third frontal, the internal surface of left frontal lobe more than the corresponding part of the right, and sparing the upper portion of both the ascending frontal gyri and part of the left postero-parietal lobe. Cerebral ventricles large, their ependyma thickened, considerable intra-cranial serosity.

F. H., Sergeant 17th Bge. Royal Artillery, admitted March 17th, 1875, then aged 31 years, service 13½ years, married.

History.—This was stated to be the first attack of mental disease, and to have become marked at the end of 1874, but the disease had been existent and insidiously progressive for a long time previously. The causes assigned were “exposure to the sun and cerebral disease.” There was no record of epilepsy, or of suicidal tendency. Of his military service, four years had been spent in India. His habits had been temperate, and he was moderately well educated. There was no history of any hereditary predisposition, nor of syphilis, nor of apoplexy. He was said to have had a blow by a poker on the right side of the head many years ago—before he enlisted. While at Singapore, four years before admission into Grove Hall, he was a good deal exposed to the sun. Two years later, when in Ireland, he was reduced from the post of Sergeant-Major, and this was said to have preyed upon his mind, and his wife stated that since that date he had been much altered in manner and disposition, having become sullen and taciturn. He had suffered from headaches since about the same period, and these affected him so severely in June, 1874, that he

was obliged to enter the regimental hospital, but he performed (!) his duties up to Oct. 1874, when he proceeded to Gibraltar with his battery. Whilst there his eccentricities became more marked, his memory quite failed him, and his speech became thick and hesitating. Said to have "softening of the brain," he was then sent home to Netley.

His father, however, informed me (W. J. M.) that the patient had a sunstroke several years ago in India and was carried, insensible, into hospital—that he was never the same again—was stupid, dazed, and occasionally, say every two or three weeks, was violent, and would chase his wife out of the house with a bayonet—that his fellow N. C. officers, with kindly intent, concealed his vagaries from the superior officers, but that at times, whilst at Sheerness and before setting forth to Gibraltar he was wild—"quite a maniac," as his father expressed it. The latter added spontaneously, that he thought the patient had really been insane for three or four years, and that when he lost his rank (in 1872 or 3), because he improperly signed some military document, he was in reality too confused to understand the nature of its contents.

The medical certificates, on which he was transferred to my care, testified that this patient had impairment of perception and of the reasoning faculties, and of speech, failure of memory, especially as to recent occurrences, apathy, inability to take care of himself, or to connect ideas of the simplest kind; that in giving any explanation he forgot words, lost the normal connection and association of ideas, and became incoherent.

State on admission.—(March, 1875.) Height 5ft. 10in., weight 162lbs.; pupils equal, scarcely sluggish, the right one irregular. The muscles of the face were jerked spasmodically and twitched, even occasionally when he was not attempting any voluntary movement, but this condition was increased when the tongue was protruded. There was also tremor cōactus of the pronators and flexors of both forearms and hands, one or both being affected, and to a degree which varied from moment to moment. The tongue was jerked in the same rhythmical manner when it was protruded. The facial expression was less fatuous than might be expected. The speech was slow, interspersed with long pauses, quasi-stammering, the words at times being tremulous and broken, but for the most part elided in portions and sometimes uttered explosively. At other times speech was very much better. During speech tremors and twitching of the facial muscles occurred. The tactile sensibility of the skin was very generally diminished, at least this was noted on the neck, chest, abdomen and extremities.

The cardiac impulse was very obvious near the epigastrium, less so near the left nipple, and was very strong and wide. The first sound was clear and full, and a cardiac bruit heard over the front of the chest appeared to be diastolic. The pulse was full and slightly jerking.

A psoriasis spot was seen on the left arm, a wart on the buccal mucous membrane, there was no scar on the penis; the patient said he had had "venereal" in 1862.

The patient was incoherent, childish, quiet, obedient, demented, extremely amnesic. His countenance was always either smiling or void of emotional expression.

Rx. Potassii iodid. grs. viii, et hydrarg. perchlor. gr. $\frac{1}{16}$ ter in die.

May 24th. Increase each dose of iodide to grs. xii.

June 28th. Omit perchloride of mercury from mixture.

Aug. 30th. Increase potassic iodide to grs. xx, three times a day.

Oct. 3rd. Resume the mercurial perchloride. Nov. 10th. Omit the mercurial perchloride.

To return. June 19th. The fingers of the right hand were held straight and, as he stood, jerked the forearm rhythmically from the side of the thigh, and there was a similar but slighter rhythmical spasmodic tremor of the lower lip. The tongue was tremulous on protrusion. The speech was broken and shaky, and during it there was slight trembling of the labial and facial muscles. Speech, moreover, was mumbling, pausing, hesitating, and this condition was succeeded by explosive utterance. The "symptomatic" paralysis agitans occasionally affected the left

upper limb also, particularly the left forefinger. The tremor and the dementia had increased of late.

June 23rd. Was very confused and dazed, and apparently suffered from headache. The pulse was 114 and jerking, the head warm, the pupils equal and acting. The patient, very paretic and helpless, was ordered to bed and to take his mixture every three hours (96 grains of K. I. in 24 hours).

June 28th. Better, pulse 102. The tremor coactus now affected the right forearm only. (Omit Hg. P., continue frequent doses of K. I.) July 18th. Much improved.

Aug. 3rd. Much as usual. Speech indistinct, broken and shaky. There was slight tremor coactus of the fingers of the right hand, ceasing temporarily when attention was drawn to it. The movements thereof varied from 60 to 80 per minute, pulse 118, soft, pupils equal. Mental perception was slow, attention and comprehension enfeebled. There was simply a disintegration or dying out of mind. (30th K. I. grs. xx. t.d.)

Oct. 2nd. Again very heavy and confused (add Hg. P. to the K. I., and take every four hours). After this date he often complained of his head, and on these occasions was unusually stupid, and occasionally wetted himself. The paretic state and the jerky tremulousness also increased again, but he could still walk, though only slowly and unsteadily. Later on, there was some "iodide coryza." To summarize the notes, it may be said that during October and the beginning of November he remained much the same, gradually failing, but varying in state from time to time, the general paresis seeming to affect the right limbs more than the left, especially as revealed in the gait, which was awkward and somewhat stiff. (Hg. P. omitted.)

Nov. 9th. Sudden dextral hemiplegia came on without convulsions, or loss of consciousness, or falling, or syncope, but the patient was found to be more dull, confused, and fatuous, and the face wore an expression of "shock." He swallowed badly, and vomited several times during the day. Temperature, right axilla, 98° 9'; left, 98° 7'; pulse varying from 60 to 70, full, soft, rather quick; respiration normal. (Mist. K. I., 4tis. horis sumend.)

Nov. 11th. Temperature, right axilla, 99° 3'; left, 99° 1'. Slight conjugated deviation of head and eyes to the left. Mental state as on 9th and 10th.

Nov. 13th. Temp., right axilla, 98° 8'; left ditto, 98° 6'. Same heaviness and fatuity.

Nov. 16th. Temp., right axilla, 98° 3'; left ditto, 97° 4'; pulse 84, very soft, full, quick and bounding. Fluids were swallowed well, the tongue could not be protruded. The right hemiplegia and mental state were much the same.

Nov. 19th. Some dysphagia. The right orbicularis palpebrarum did not resist being opened so much as did the left. Later on, some spasm affected the mouth, which was opened more widely at the right commissure, and the tongue was convulsed;—fibrillar tremor also affected the lower fibres of the right orbicularis palpebrarum. This was followed by general spasmodic jerking of the masseters and other muscles of jaws on both sides, and by involuntary grasping, forward movements of the left hand.

Nov. 21st. Temp., right axilla, 97° 7'; left ditto, 96° 7'; pulse 74.

Nov. 23rd. Had a number of spasms, limited to the right side of the face, every day, and lasting from $\frac{1}{2}$ minute to 10 minutes. 25th. Temp., right axilla, 98°; left, 97°. Could reply only by an unintelligible monosyllabic mumbling. 26th. Temp., left axilla, 98°.

Nov. 27th. Synergic contraction of head and eyes to the left. Right face paliaed and flattened, right arm paliaed, but resistant to passive motion. The mouth gaped, then spasm of the lips was succeeded by tremor extending to the muscles of the cheeks, then the mouth was in spasm, the tongue protruded and bitten, next the mouth was agape and the tongue retracted;—and throughout there were wavy fibrillary tremors of the face, nostrils, and lower eyelids, especially on the right side,—also of the right platysma. Then the tremors increased and spread and the right corrugator supercilii was involved thereby before the left. The spasmodic twitching tremor was intermittent. Temp., right axilla, 99° 6'; left ditto, 99° 2'. Fed by oesophageal tube and pump.

After this day the spasms subsided, the right hemiplegia gradually lessened, the power of movement returning first at the proximal parts, the patient grew brighter again in appearance and less fatuous and helpless, the pulse varied from 102 to 108, and power returned, so that by Jan. 1876 the patient was able to walk a little by himself, and to be regularly exercised by an attendant.

Jan. 29th, 1876. Sudden right hemiplegia occurred; ordered to bed, which he did not again leave during life.

Feb. 6th. Was drowsy, swallowed badly.

Feb. 12th. The pupils were equal, wide, and sluggish; the patient swallowed badly; his knees were somewhat flexed and contracted. In the evening the face and limbs were cold; the pulse was slow, and there was vomiting (stimulants, hot bottles to feet).

Feb. 18th. Vomiting, cold face and limbs, moaning, pulse 44, soft, of medium size, heels drawn up to the buttocks. The arms were slightly contracted, especially the right. The right limbs resisted passive motion less than the left. The conjunctivæ were injected. This condition continued until death on Feb. 14th, 1876.

Necropsy, 38 hours after death. Body well-nourished. At the left side of the forehead was a scar, adherent to a smooth depression in the bone. Calvarium capacious, thick and dense, the portion removed weighing 21½ ozs.

Dura-mater, somewhat too adherent to the calvarium, especially at the base in the left middle *fossa basis cranii*; rather thickened and hyperæmic. Sinuses gorged with dark fluid blood.

There were marked basal interlobar adhesions, and at the base the arachnoid was thickened and unduly opaque. Over the whole superior and lateral (external) surfaces of the frontal and parietal lobes, over the upper half of the temporo-sphenoidal lobe, the anterior part of the occipital, and the anterior two-thirds of the internal surfaces of the hemispheres, the membranes were extremely thick, opaque and adherent; the sulci of these parts were full of serum, and the membranes stripped off with great difficulty. These changes were most marked over the upper surface, and were symmetrical in their external appearance. Shreds of pia-mater remained in the somewhat wide and rounded sulci.

Adhesion and decortication affected more or less the prominences of the gyri on the superior and external surfaces of both cerebral hemispheres, affecting the parietal lobes most, and both temporo-sphenoidal and both occipital lobes slightly. The adhesions were almost absent at the anterior part of the frontal lobes; especially were the anterior halves of the second and third frontal gyri comparatively free.

By this change also the posterior part of the right third frontal gyrus was slightly more affected than the corresponding part of the left. The inner surface of the left frontal lobe was more affected than the same surface of the right. The upper part of the ascending frontal gyrus on each side escaped partially. The left postero-parietal lobule was not much affected by the same change, nor indeed was the corresponding part of the right side. The inferior surface of both hemispheres was free from the alteration in question.

In the right cerebral hemisphere the grey cortical matter was pale, and only a few minute vessels were visible therein to the naked eye. Its deeper layers were rather firm: this firmness extended through the fronto-parietal region, was very slight in the occipital, and absent in the temporo-sphenoidal. The strata were not very obvious, they were best marked in the parietal convolutions, and only faintly marked in the occipital. The grey matter was of fair depth, but was, proportionally, of unusual depth in the occipital region. Its colour externally was a slaty grey; internally of a dirty whitish and faintly yellowish hue.

In the left hemisphere the grey cortical matter had much the same appearances as that of the right, but the colour was rather paler in the frontal region. Its consistence was more increased than on the right side. The grey matter of the lower part of the left, and to a less degree of the right, frontal lobe was firm, very pale and homogeneous in appearance. Its stratification was best marked in the ascending frontal gyrus.

The white cerebral substance was of increased consistence. Its puncta cruenta

were very small, and its vascularity lessened, the colour being white with a very faint pink tinge, and paler in the left than in the right frontal region.

The lateral ventricles were very large and contained a considerable amount of serum; their ependyma was thickened. Grey commissure present. Basal ganglia rather pale, of ordinary consistence.

Grey matter of cerebellum pale. Pons Varolii and medulla oblongata slightly firm.

Left hemisphere, 23½ozs.; right hemisphere, 24½ozs.; cerebellum, 6½ozs.; pons Varolii and medulla oblongata, 1½ozs. Six-and-a-half fluid ounces of serum and blood escaped. No thrombosis, embolism, or strictly local softening were found.

Thorax.—*Heart*, 12½ozs. The aortic semilunar valves, thickened and unduly opaque, were rough and irregular, especially on their ventricular aspect, and one appeared to be slightly incompetent. The mitral valves were large and thickened; the other valves healthy. In the arch of the aorta were some atheromatous patches. The left ventricle was contracted, it was also hypertrophied.

Lungs.—Left lung, 29½ozs.; right lung, 43ozs., emphysematous in front; congested, oedematous, and the site of some hypostatic pneumonia posteriorly.

Abdomen.—Right kidney 6ozs., left, 7½ozs.—*Spleen*, 4½ozs., some old adhesions between the capsule and surrounding structures.—*Liver*, 54½ ozs.

Remarks.—1. In this instance certain lesions and symptoms apparently had their starting-point in insolation. The moral cause suggested by some of the friends for the malady seemed to have been brought into operation after the prodromes of the disease had occurred. The blow on the head occurred at too remote a period to merit consideration here.

2. As to why the unilateral convulsions and spasms should have been mainly of the right side, this was not very obviously accounted for by differences in the distribution of the adhesions over the two hemispheres. The left hemisphere, however, was the one more diseased, its atrophy being greater, and its frontal grey cortex being paler and firmer than that of the right hemisphere.

3. The right hemiplegia was too persistent to be purely epileptic hemiplegia, but there were no obvious changes to account for it, save the differences just mentioned in the condition of the two cerebral hemispheres.

4. The brain was naturally a very large and heavy one.

5. There was an entire absence of characteristic syphilitic changes after death, though some of the changes (e.g., the meningeal) might have been syphilitic.

CASE XIII.—In the *Journal of Mental Science* (January, 1876, p. 567), I have already described this case in detail, and have appended a lengthy commentary. A very brief summary, therefore, will suffice here.

Extreme maniacal excitement, incoherence, the patient noisy, destructive, violent; grandiose delusions. Intense irritability and furious outbursts of anger, the patient threatening, or even attacking others, proud and haughty in bearing. Auditory hallucinations: early epileptiform convulsions: insomnia.

Later, tractable, quiet, dull, inert, with failing mental powers. Motor signs of general paralysis at first comparatively slight: great motor restlessness. Later, right unilateral epileptiform convulsions, followed by right hemiplegia and temporary aphasia. Finally, bedridden, recurrence of epileptiform convulsions and of right hemiplegia. Death in the status epilepticus.

General diminution of consistence of encephalon. Left hemisphere 2½ozs. less than right. Meningeal changes more marked over left side. Left hemisphere the softer; and the grey cortical matter, atrophied on both sides, was more so on the left, and especially on the superior and external surfaces. Adhesion and decortication much more marked in left hemisphere, and observed especially over the posterior part of the parietal lobe, and posterior part of the frontal, but of wide distribution. Grey cortex of ordinary hue. Medullary substance hyperæmic. Marked granulations of fourth ventricle. Meninges thick and hyperæmic over pons and med. obl., and these parts hyperæmic.

GROUP IV. (see p. 182).

CASE XIV.—Attack began with acute maniacal symptoms, and then, or very soon after, were exalted delusions. Frequent paroxysms of dangerous excitement, violence, destructiveness. Later on, the same maniacal excitement, with absurd and contradictory exalted and extravagant delusions, the patient sometimes elated, and sometimes irritable and dictatorial. Hallucinations of sight and hearing. General shivering, tremulousness, excessive motor restlessness. The impairment of speech masked by the state of agitation. Later on, subsidence of excitement, appearance of motor signs. Later still, hypochondriacal delusions, lachrymose depression, and, no doubt, morbid hypochondriacal sensations. Death after violent epileptiform convulsions lasting 40 hours.

Great venous engorgement of meninges from mode of dying. Other meningeal changes ordinary. Brain hyperæmic, of diminished consistence. Some atrophy of grey matter in the anterior regions. The right cerebral hemisphere 2ozs. less in weight than the left. Adhesions much more decided and extensive over the right than over the left hemisphere, and in their more marked degrees were confined to the lateral surfaces and to the anterior extremity of the superior surfaces. Ependyma ventriculorum (4th and lateral) thickened and granular; cerebellum hyperæmic; its meninges the same, and thickened.

H. R., Private 4th Hussars. Admitted Feb. 26, 1876, then aged 36 years, 18½ years' service, married. Neither epileptic nor suicidal.

History.—This attack of mental disease was said to be the first, and to have lasted from January 12, 1876, and the cause assigned for it was his reduction to the ranks. He had previously been under care and treatment in the military hospitals at Canterbury and at Netley, into the latter of which he was admitted only eighteen days before his transfer to Grove Hall. There was no record of epilepsy or of palsy in his medical history sheets, and no hereditary tendency was known. He had then recently been reduced from the position of troop sergeant-major to that of private, and sentenced to six months' imprisonment, for deficiencies in his accounts. This had preyed much upon his mind and was believed to be the exciting cause of his mental disease, which, so it was said, began suddenly on January 12, and presented symptoms of "acute mania."

His wife denied that he had been addicted to any sexual or alcoholic excess, or that he had misappropriated the missing money to his own use.

Upon his leaving Netley for Grove Hall it was reported of him to this effect: "He is eminently irrational in his conversation and manner; has delusions of being possessed of immense wealth, and exalted ideas of his connections and importance. He is subject to frequent fits of excitement, during which he offers violence to the attendants and patients; is most destructive in his habits, and by his noise disturbs the inmates of the hospital. He is a dangerous lunatic." The medical certificates also referred to his marked incoherence and delusions of wealth, to his exalted ideas of self-importance, and to his outbreaks of dangerous and destructive violence.

State on admission.—Rather short and muscular, pupils equal, cicatrix in left groin, pulse ninety and full, viscera normal.

For several days he was either elated, or irritable and dictatorial, very excited, restless, noisy, and threatening, giving utterance to delusions; such as that he was possessed of extraordinary strength, that he could travel many miles in a minute, had been forty-three years in the army, and was the first man in it. The head remained heated, the patient flushed very readily. He had a bilious sallow hue of the skin, a dirty-furred tongue, a frequent soft pulse. The excitement was constant—the insomnia complete, indeed no sleep was obtained until the fifth night. Ordered warm baths, cold to the head, aperients, and night draught of chloral hydrate. The motor signs of general paralysis were not present during this period.

Subsequently, when the excitement had subsided in some degree, the tongue was slightly tremulous and twitching, and the whole muscular system was agitated by slight movements, almost precisely as if the patient was shivering slightly from head to foot from the effects of cold, though he was manifestly warm and comfortable, and, on the other hand, was not the subject of febrile rigor. If the eyelids were closed the pupils *dilated* when the lids were again opened. When H. R. was quiet his words were at times slightly clipped. The cheirography was fairly natural. He abounded in extravagant delusions. "Has millions of money and it doubles every minute; has hundreds of thousands of wives and innumerable children; is extremely strong; gives the schools £10,000 a day; has been married and in the service forty-three years, but is only thirty-five years old." He averred that at the moment of speaking he was riding in a railway saloon carriage with his wife, and mentioned to me the stations as they arrived at them. "Now we are at Suez," he said; and next moment, "we are at Paris;" and the moment after, "at Australia."

There were hallucinations of sight and hearing, the motor restlessness was extreme, as well as was his interference with surrounding objects. In his excessive restlessness and violence he fractured a metacarpal bone by striking his fist against the wall of his room. Each night a warm bath was administered at bed-time, and followed by a draught of chloral hydrate gr. xl.

By March 4th he had become quiet, eating and sleeping well, but the shivering tremulousness and the mental condition were unchanged.

March 27th. Had continued to sleep well since, and was now quiet and exercised regularly. Sometimes he was weeping, sometimes elated, and he had extraordinary and absurd delusions. At this date the mouth was somewhat drawn up towards the left side, the pupils were small, irregular, rather sluggish, and the right dilated partially when the closed lids were raised. The tongue and lips trembled much when the tongue was protruded; utterance was clear, but there was a faint undertremor of voice like that of a person who is shivering, and the universal shivering-like tremor continued at times. He now had incipient left ophthalmoma. There were no signs of increased arterial tension. To omit the night draught of chloral, and to take a mixture of extract of physostigma and perchloride of iron.

April 6th. Was occasionally restless, or showing emotional distress. On the 4th, he had dashed his fist through a pane of glass.

On May 30th he tripped, fell, and fractured the right fibula.

The notes of June refer to his extravagant delusions.

July 24th. He refused his food on this day. He said of himself, "I can't open my mouth," "I'm nearly dead," "I can't eat," "Nothing comes from me." Then he wept. The pulse was small, compressible, 74, but about two minutes afterwards it had risen to the rate of 104 per minute. The heart-sounds were of ordinary character. The hands were cold. The tongue was flabby, and the breath foul. By the time these notes were made the patient added: "I can't pass water," "I'm as mad as a March hare." He was ordered an aperient draught, which he swallowed after much coaxing and reassurance, but, immediately, he bellowed out that "he was dead or nearly so," "had nothing in him to pass," and "was a duffer."

The bowels were moved freely, and on the next day (25th) he was cheerful, was reading, and had extravagant delusions, saying that "he had £1,100,000 a minute, and constant telegrams from his heavenly Father." The pulse was now 102, soft, the head cool, tongue clean, breath sweet, and pupils as usual.

July 26th. Was again depressed and refusing food, but was silent.

During *August and September* he frequently expressed delusions of a hypochondriacal nature, such as those mentioned above, and was at times depressed and weeping. Occasionally, however, he gave expression to extravagant delusions, though without any indication of pleasure or elation in his tone or countenance. The pulse, frequent, soft, and rather small, latterly became of about normal frequency, and at a later period, again slightly too frequent, and the arterial tension was diminished. On August 16th the physostigma had been omitted, the iron being continued.

Oct. 1st. He was now less hypochondriacal, and was stouter and stronger. The face was of a dull reddish sallow hue. Epistaxis occurred spontaneously. The motor indications of general paralysis in the speech, tongue, lips, etc., continued.

Oct. 13th. He was sitting down at 4 P.M. talking quietly to an attendant, when suddenly he uttered a cry, stiffened, and passed into violent convulsions. Immediately I visited him and found him in the *status epilepticus*. Each fresh convulsion that supervened on the continuous violent clonic spasms, began with stiffening and straightening of the right upper extremity, with violent spasm drawing the face head and eyes towards the right side, but almost at once making way for general clonic convulsions, the face being swollen and turgid, and profuse generalized perspiration coming on. When these general clonic convulsions subsided there were left unilateral spasms, the head being drawn to the left, but the eyes upwards and to the right, while the right upper extremity was paralyzed. The pulse and respiration were rapid. The severity of the convulsions lessened in two hours, but epileptiform fits recurred from time to time, beginning with tonic spasm, mainly of the right side, and then becoming general. The pupils were sluggish, of medium size. The respiration rose to 52, and again fell to 30, and to less, per minute. In the evening, and continuing all night, were spasms, mainly of the left limbs and of both sides of the face, especially the right. The bowels were cleared out by enema of magnesian sulphate, then several enemata of chloral hydrate were given,—also milk, brandy, and potassic bromide, and inhalations of amyl nitrite.

Oct. 14th. He still had twitching of the arms, also, to a slight degree, of the legs, especially the right, and well-marked twitching of the face on both sides. The mouth was jerked somewhat to the left, the head to the left, but the eyeballs upwards and to the right, and there were frequent spasmodic jerkings of the upper limbs, especially of the left, and of the thighs. Reflex movements were more impaired in the right lower limb; reaction to painful sensory impressions failed in both lower extremities, paralysis affected all the limbs, particularly the left. The respiration was 27 and stertorous. The patient had been more or less comatose throughout.

At 1 P.M., temperature, 99·5°: respiration 30, laboured. Had twitches of the face, arms and legs, especially of the right, which were persistent. Had had five protracted severe convulsions this morning. Then, in a fit, which came on whilst this note was being made, the head and eyes turned to the right, and after it was over they turned to the left again—their previous position. Some abdominal tympanitis; lungs congested; bowels freely moved; catheterization necessary.

Later still in the day, convulsions continued. Immediately were twitches, especially of the face, arm, and trunk, the last-named being throughout markedly affected thereby. At midnight (Oct. 14–15) the pulse was 125, respiration 45. The fits then taking place began with spasm of the right side of the face and of the right limbs, and became general. Both thoracic limbs were paralyzed. During the night there were fifty or sixty seizures of epileptiform character, which continued until death at 8 A.M. on Oct. 15th, 1876.

Necropsy, thirty hours after death. Body well nourished. *Head*.—The calvarium was thin, and soft, its diploe moderately injected, weight of portion removed 130m. Sinuses gorged with dark fluid blood and clot.

The dura-mater was congested and apparently slightly thickened. The soft membranes (pia-arachnoid) presented considerable venous congestion. They were thickened, unduly tough, and showed a faint milky opacity over the whole of the superior and lateral surfaces of the brain, except the posterior two-thirds of the occipital lobe. The temporo-sphenoidal meninges suffered much less. The same meningeal changes were also noticed in a slight degree over the internal surfaces of both hemispheres, in the parts of them corresponding to the frontal and parietal lobes. The pia-mater was slightly infiltrated with serum.

The membranes stripped off with some difficulty; and from the prominences of some of the convolutions the superficial layers of the grey cortical matter separated along with the meninges. This adhesion was more marked in the right cerebral hemisphere than in the left, and in this right hemisphere it was most marked over

the lower half of the parietal lobe, the lateral surface of the temporo-sphenoidal lobe, the lower part of the right ascending frontal gyrus, the right third frontal gyrus; and, to a less marked degree and extent, over the anterior and posterior extremities of the middle frontal gyrus, leaving an intermediate oasis unaffected, and over the whole of the superior surface of the first frontal gyrus. It was observed slightly on the orbital surface, and on the internal surface, but the occipital lobe escaped, with the exception of the anterior part of the third occipital gyrus. Thus a large band on the posterior two-thirds of the vertical portion of the right hemisphere escaped this change, and the breadth of this oasis was from two to three inches at different points, and it was limited anteriorly by the first frontal gyrus.

The adhesive change was much less advanced in the left hemisphere, but had a somewhat similar distribution, except that the superior surface of the first and second frontal gyri escaped, the orbital surface, however, being as much affected here as on the right side. The temporo-sphenoidal lobe, moreover, was equally or more affected on this side, and the third frontal gyrus was considerably implicated.

Numerous vessels were apparent in the cerebral grey cortical matter, which had a faint lilac hue, and which seemed to be atrophied in the anterior regions.

It may be added that in portions of the right first frontal gyrus, examined in the fresh state with the microscope, a few of the large pyramidal nerve-cells showed some granular degeneration, but others were healthy. Numerous nucleated cells, and leucocytes were seen, and one deposit of blood-pigment.

The whole brain was of diminished consistence.

The white matter was hyperæmic, had numerous puncta cruenta, and was decidedly pasty.

The fornix and grey commissure were soft. Considerable serous fluid was found in the lateral ventricles of the brain, the lining membrane of which was thickened and granulated. The corpora striata were of a dull lilac, the optic thalami of a mottled lilac hue.

The left hemisphere weighed 23½ ozs.; the right, 21½ ozs.

There were slight toughness and opacity of the membranes of the cerebellum, the grey cortical matter of which was slightly hyperæmic, especially on the right side near the median line. Immediately beneath the posterior extremity of its inferior vermiform process was a slight pia-matral hæmorrhage. Weight of cerebellum, 5½ ozs.

Pons Varolii and medulla oblongata, moderately hyperæmic. Weight ¾ oz. Lining membrane of fourth ventricle slightly granular and thickened. Serous fluid from cranial cavity, fl. oz., 1¼.

Thorax.—One oz. pericardial fluid. *Heart*, 11½ ozs. The muscular substance of the heart was of a dull purplish and yellowish hue. A few atheromatous patches were seen in the arch of the aorta, and in both coronary arteries were numerous, slightly raised, irregular, yellowish-white, and apparently atheromatous patches, giving a somewhat corded and beaded character to the arteries.

Right lung 31½ ozs. *Left lung*, 26½ ozs.; posterior congestion of both, and slight hypostatic pneumonia of right.

Abdomen.—*Stomach*, distended by flatus. *Spleen*, 2½ ozs., firm, and rather pale. *Left Kidney*, 4½ ozs., slightly lobulated; its capsule slightly adherent. *Right Kidney*, 4½ ozs. *Liver*, 57½ ozs.

Remarks.—1. Here were found the ordinary post-mortem indications of general paralysis, but much more adhesion and decortication in the right than in the left hemisphere, and the right hemisphere was 2 ozs. below the left in weight.

A wide vertical band, comprising part of the superior aspect of the right hemisphere, was free from adhesive change, except over the first frontal. This change, less in degree and extent in the left hemisphere, had a somewhat similar distribution to that in the right.

4. The unusual distribution of the adhesive change, and of the cortical changes associated therewith, might be thought to have had relation either to:—

(a.) The comparative absence of the physical motor signs of general paralysis

in the early stages. This, however, I do not think to be the case, the early absence of these signs was, I think, due to the "masking" effect of the extreme maniacal furor then present.

Or (b) one might try to trace some relation between the points of the cortex specially irritated by the adhesive change, and the distribution of the convulsions which cut short life. This distribution was variable, being mostly on the right side during the epileptiform convulsions, but more on the left side during the spasmodic twitching of the intermediate period. Now, if we transfer the results of experiments on the brains of monkeys to the elucidation of these convulsions and these spasmodic phenomena, we are constrained to say that no decisive relationship can be proved in this case between the course, march and distribution of the convulsions and spasms, and the distribution of the adhesive change. Yet was there probably an intimate relation between the two.

CASE XV.—Insidious in onset, patient eccentric and peculiar for a long time before decided mental disease was recognised. Exalted delusions, maniacal excitement, destructiveness, filthy habits. Later on, usually elated, joyous, happy, and expressing the most extravagant, exalted, changeable and self-contradictory delusions, and the most benevolent and generous intentions. Later still, the exaltation alternated with an ever-increasing and supplanting condition, in which the patient was depressed, hypochondriacal, morose and querulous. The language became abusive, threatening, and fouler than ever. Finally, dementia had advanced apace, the mind was inclouded by hypochondriacal feeling, and the patient, usually silent, was of dolorous, woebegone appearance. Motor signs of ordinary character; finally much dysphagia and speechlessness. Severe recurring epileptiform convulsions, followed by left hemiplegia, conjugated deviation of head and eyes to right, and a higher sinister temperature; latterly low temperature. Finally, frequently recurring convulsions, an apoplectiform state, and the third degree of left hemiplegia.

On right cerebral hemisphere very recent meningeal hæmorrhage. No adhesion to cortex. Right hemisphere, 1½ozs. the less in weight: and its grey cortical substance softer in the frontal region in this than on the left hemisphere. Meningeal changes moderate. Ependyma ventriculorum thickened and granulated.

G. S., Private 52nd Regiment. Admitted Dec. 9th, 1874, then aged 35 years: service 16½ years, single.

History.—This was said to be the first attack of mental disease and had been previously treated at Gibraltar and Netley; its cause was said to be uncertain. The patient was said to have had an attack of "epilepsy" in June 1866 but none since. He was of intemperate habits.

Mental symptoms were stated to have become marked at Gibraltar in August 1874, whilst he was undergoing imprisonment, but he had been considered by his comrades as eccentric in his habits and peculiar for some time before. Admitted into Netley Hospital on November 6th he, while there, expressed exalted delusions as to wealth and power, and was filthy and destructive in his habits. The medical certificates furnished at Netley spoke of occasional excitement, general incoherence, exalted notions as to wealth and power, dirty habits, tendency to destroy clothing, and "paralysis of the insane." They specified delusions such as that he could speak seven languages, was fitted to be a railway military engineer, and could get £500 by writing an order for it. They specified also, that his letters were incoherent, and full of the same delusions.

State on admission. Physical.—Weight, 167lbs, fairly built and nourished, depression at vertex of cranium. Wide area of cardiac percussion dulness, forcible cardiac impulse, and intermittent irregular action, pulse small, weak, intermittent, 72. Other viscera healthy. Some very slight irregularity of ordinary character on the shins, and cicatrices on the legs. The patient said he had had syphilis, but

there was no history of it, and no proof of its existence then. The condition of the speech, and the tremulous twitching of the lips and tongue, were those usual to general paralysis in the early stage.

Mental state.—His conversation was rambling, incoherent and trifling, and he gave the most varied and contradictory accounts of his past life, or of his wealth, and often expressed the most benevolent and generous designs.

At times he was much excited, at others, in good-humour, quite joyous and laughing heartily at trifles. He thought he had unlimited wealth, saying "I am possessed of millions of millions." He gave orders for immense quantities of various articles and directed that the following should be sent to him immediately: "25½ lbs. of tobacco, half a dozen of Eau de Cologne, four concertinas, a paper shirt and a paper cravat, 60½ dozen of pocket-handkerchiefs, a field marshal's uniform and bâton, 1,009 boxes of hams, 26,000 lbs. of currants, a stage, and a carpenter." To pay for all this, he said "Draw on Cox & Co. to amount of £150,000 or more if it is necessary." He also stated that God sent him all the money, that he had large estates and kept, in his stable, hunters, one of which had won "the Derby." He said "Before I went into the army I was a physician to Guy's Hospital with a salary of £10,000 a year." Again, "The colonel, major, and doctor all conspired against me, and I will get them all hanged." "My father made all the clothing for the army, my mother was a lady in her own right and took in washing." Then he contradicted his previous statement, saying: "Before I 'listed' I was an undertaker, kept a donkey, washed clothes, pawned, and washed them again, for a living." "In 1800 I was sixteen years old, went to America as a farmer, and sailed round the world." R. Potassii iodid. and ammon. carb.

March 20th, 1875. Delusions much the same. To omit the above mixture and to take perchloride of iron, and, on May 6, ordered a night draught of potassic bromide.

By June 1875 he was quiet, and his delusions were partially in abeyance. Now and then he declined food. Palpitation and dyspnoea on exertion, pulse 90, intermitting 1 in 20. The manual grasping power was lessened. The night draught was omitted, and the mixture of potassic iodide was resumed, with the addition of hydrarg. perchlor.

Dec. 9th, 1875. He sometimes destroyed clothing, and then said other patients did it. He had now for some time been querulous, morose, grumbling, sullen, profane. His favourite saying, that "he was happy," was rarely heard now. He, however, said he was "a general and had ten shillings a day as pay." His memory was bad, speech somewhat hesitating and tremulous, and tongue not kept protruded well. The slight cardiac bruit had disappeared. Omit the mist. K.I. &c., continue the mist. Ferri. Weight 176½ lbs.; gain since admission, 9½ lbs.

Jan. 3rd, 1876. On the preceding day the mouth had suddenly been drawn up on the right (?) side, and the patient became heavy and stupid. On this day there was moderate left hemiplegia, and semi-coma, but the head and eyes were turned to the left (*sic*); later, however, they were turned to the right, and the pupils were dilated. At midnight the right limbs were cold or chilly, and the left were recovering their motor power.

Jan. 4th. Same state. Right limbs, feet cool; the left warm. Temperature, right axilla, 100·2°; left, 101·2°.

Jan. 5th. Occasional convulsions. Left hemiplegia persisted for some time, and was very marked (second degree). No albuminuria; the heart's action was intermittent.

Jan. 8th. Convulsive seizures, both day and night, affecting principally the left side of the face. Afterwards, the head, and eyes, and face, were drawn to the right, and left hemiplegia was marked, and was of the common form. The right hand felt cold, the left warm.

Jan 9th. At visit, left hemiplegia marked, no fits since the night. Temperature, right axilla, 95·8°. The thermometer had risen to 96·6° in the left axilla, when a convulsive seizure came on, to be succeeded by others. The description of one will suffice: 1st stage, (a) closure of left eyelid, by orbicular muscle, with fibrillary

tremor; (b) to this were added spasmodic clonic movements of the mouth, also affecting the cheeks, especially the left, the eyeballs being turned upward and outward to the left:—2nd stage, slow turning of head from right to left, spasm of left face increased, and that of left eyelid relaxed, the tongue was actively convulsed, but not protruded, the muscles of the neck became involved;—3rd stage, extension, rigidity, and rapid fibrillary quiverings of the muscles affected. The left arm now moved for the first time, it was rigid and raised, the wrist and elbow somewhat flexed; the occiput boring backwards, the mouth widely open, chin to left, left lower limb rigid;—4th stage, momentary rigidity of right limbs, and general clonic convulsions affecting the right more than the left arm. Then the condition of the second stage returned, and with it tremulous movements of the left limbs. A few minutes after, temp., left axilla, 95.5°.

Jan. 10th. No convulsions since. T., right axilla, 96.2°; left, 96.5°.

Jan. 11th. Only slight convulsions since. Hemiplegia continued, the patient could reply, T., left axilla, 97°.

He made a marvellous recovery from the condition above described, after having been supported by nutritive enemata for several days. In Feb. up and about again.

June 25th. Confined to bed, feeble, much dysphagia, and often an inability to speak. When he did speak the language was often abusive, foul, and disgusting. The patient, drowsy and stupid, waved his hands about. The urine was occasionally retained and evacuations were passed under him.

July 11th. Feeble pulse, faint systolic murmur at right side of cardiac apex, cool skin, foul breath, refusal of food. Patient was dull, unable to speak, looked dolorous, often stared vacantly, or pointed as if to imaginary objects. He had recently been affected with left othematoma, and had been up and about again.

He became more depressed, hypochondriacal, morose, and querulous; dementia was also marked; the motor power improved.

Sept. 7th. During the past night the attendant noticed him breathe stertorously, and have muscular twitchings, distorted features, rolling eyes, clenched fists, and flexed limbs. "A convulsion" at 5 A.M. was reported, and afterwards there was left hemiplegia, with conjugated deviation of the head and eyes to the right. The mouth was drawn to the right, and gaped at the right commissure of the lips. Pulse 92, soft; respiration heavy and laboured; temperature, right axilla, 102°; left do. 102.5°; skin hot and dry; lungs inflamed posteriorly. Patient dull and drowsy.

Sept. 8th. Had a general tonic convulsion, after which the hemiplegia and coma were more marked, and the respiration stertorous.

9th. Still, marked hemiplegia, conjugated deviation to right, stertor. Temp., right axilla, 99.8°; left, do, 99.6°. Patient almost unconscious.

10th. Sensation very much blunted, deglutition difficult, sensori-motor activity lessened on the left, the paralyzed, side. Head and eyes turned to the left. Patient comatose. Enemata, chloral hydrate. During the day there were twenty-four convulsions, either general, or mainly confined to the left side of the face, besides spasms of the left face. Catheterization.

12th. Pulse 126, full and feeble; respiration 58, noisy and rattling; nineteen convulsive seizures took place on this day. Enemata of chloral hydrate and potassic bromide had been given. Left hemiplegia persisted. Coma deepened, and death took place at 6 P.M. on Sept. 12th, 1876.

Necropsy.—*Head.* Calvarium, easily sawn, slightly worm-eaten appearance on inner surface by sagittal suture; portion removed, 16½ozs.

The dura-mater was deeply blood-stained except at the cerebellar fossæ; attached to its inner surface was a thin, soft, almost diffuent blood clot, overlying the right temporo-sphenoidal and occipital lobes, extending along the middle fossa of the skull-base, and covering the anterior surface of the petrous portion of the right temporal bone. There was, also, fluid blood about the base of the brain in the arachnoid cavity, and the soft meninges covering the right hemisphere had deeply imbibed the blood hue. The clot was quite recent, the source of hæmorrhage was not obvious. The dura-mater was not unduly adherent, and was of ordinary thickness and consistence.

There was faint milky opacity of the arachnoid over the superior and external surfaces of the frontal and parietal lobes. The conjoined pia-mater and arachnoid were thickened and tough, the distribution of these changes being the same as that of the arachnoid opacity; and over the same region the pia-mater was infiltrated with serum. These meningeal changes were fairly symmetrical over the two cerebral hemispheres, and the convolutions in the region just mentioned were somewhat wasted and the sulci rounded. About the base were interlobar adhesions. The membranes stripped off readily from the cerebrum, and there were no adhesions to the cortex.

On section, the surface of the cortical grey matter was very slightly stained adjoining the blood-stained parts of the meninges. The grey cortex was softer in the right than in the left frontal lobe. Both hemispheres were well supplied with blood, but the right less so than the left. The brain was, generally, of about normal consistence.

The lateral cerebral ventricles contained 3iii of serum, and their lining membrane was thickened and granulated. The basal ganglia had considerable vascularity, and no special wasting or lesion of them was found.

Weight of left hemisphere, 19½ozs; right, 18ozs. Cerebellum 4½ozs., pons and med. obl. 1oz., slightly hyperæmic, ƒ3iii serum escaped during the removal and dissection of the brain.

Thorax.—*Heart*, 10½ozs., mitral valves, coronary arteries, and aorta, atheromatous. *Lungs*: slight hypostatic pneumonia of bases; a trace of recent left pleurisy. *Left*, 34ozs.

Nothing else calling for notice. *Spleen*, 9½ozs. *Liver*, 57ozs. *Left Kidney*, 6½ozs.; *Right Kidney*, 6ozs.

Remarks.—1. In this case a suspicion of syphilitic disease was roused at first, but neither during life nor after death was its presence demonstrated.

2. This is one of those infrequent cases in which undoubted general paralysis occurs without any adhesions between the cerebral cortex and meninges. Yet the other chronic changes in the meninges, usual to general paralysis, were present.

3. In January, were frequently recurring convulsions of the left side, left hemiplegia and a higher left temperature. Later, there were recurring convulsive seizures, followed by increase of left hemiplegia, and low body temperatures. Then in September the fatal attack occurred, consisting of, (a) a slight convulsion, (b) left hemiplegia, (c) frequent convulsive and spasmodic seizures, (d) increase of paralysis and of coma.

Now in relation to these were—

(a.) Some atrophy of the right cerebral hemisphere.

(b.) Right meningeal apoplexy of recent formation. The lesion leading to the former was apparently connected with the earlier left unilateral convulsion and palsy, the latter with the final lethal seizures.

4. The final speechlessness was of mental origin; no local lesion afforded any explanation of it at the necropsy.

Case XVI.—*Insidious onset; absurd exalted delusions. Later on, these delusions still remained, and the patient was irritable, often obstinate or abusive, and evinced mingled exaltation and morosities, selfishness, and jealousy. Language often foul and profane. Early gradual loss of sight and blindness. Later on, the patient was morose, depressed, sullen, and, on occasion, stupid, heavy, confused, and stricken with left hemiplegia, and impaired sensation in left upper limb, and a lower left temperature.*

Fatal illness; loss of consciousness, sudden interference with respiration and deglutition, right hemiplegia and paralysis of left third cranial nerve; diaphragmatic respiration; cold hands and feet; earthy sallow pallor.

Right hemisphere 1½ozs. less in weight. Grey cortical matter of right side

thinner, paler, and more uniformly of slightly increased consistence than in the left hemisphere. Adhesions over the superior and lateral surfaces, especially the posterior part of the frontal, and the lower and anterior part of the parietal lobes, moderately on the temporo-sphenoidal, slight on the anterior two-thirds of the internal and inferior surfaces. Angular gyrus, postero-parietal lobule, and occipital lobe almost free therefrom. Ependyma v. thickened and granulated. Embolism of left middle cerebral artery, and embolic softening of part of left corpus striatum. "Surgical" kidney, and embolism.

H. L., Private 7th Regiment. Admitted July 21st, 1875, aged 37, service 17½ years; married.

History.—First attack, insidious in onset, and probably of nearly a year's standing at the time of admission to Grove Hall, but only under active treatment since April, 1875. Cause "unknown." The wife, however, informed me (W. J. M.) that the patient had for many years been addicted to drinking habits, especially during the six months preceding his admission into hospital in April, 1875. She also stated that in sexual matters he was decidedly of a frigid temperament, and mentioned particulars which proved the truth of her assertion. He had always been selfish, unkind to, and careless about, his wife and children, and was harsh and hostile to her when he was in liquor. He inherited a cold and selfish nature from his mother. No other family history was procurable, and there was no record of epilepsy, suicidal tendency, syphilis, or injury to the head. From April to July 10th he was treated at Dover; afterwards, at Netley.

The medical certificates on which he was admitted into Grove Hall testified to his complete irrationality and incoherence, self-satisfied complacent manner, exalted and absurd ideas of his rank, station, wealth, and physical power,—such as that he was commander-in-chief of the army, or a member of the royal family, and that he fought at Waterloo. Also to the great impairment of sight, hesitation of speech, and progressive general paralysis.

State on admission.

Physical condition.—Height, 5ft. 6in. Weight, 133lbs., cranium rather small, florid complexion, pupils wide, especially the left, both sluggish, and of irregular shape; the fundus of the eye was of hazy appearance, sight was impaired, the right eye being apparently the weaker, but he would not allow ophthalmoscopic examination at that time. The tongue was protruded tremulously, and there were then very marked tremors of the lips and face. Speech was rapid as a rule, but occasionally hesitating and stammering. The gait was slouching and wanting in firmness; the hands and fingers were tremulous and uncertain when employed to button clothing.

Heart-sounds clear, second sound slightly accentuated, pulse 102. Varicose veins in legs. He denied having had syphilis, and there was no proof of its existence, past or present.

He was restless, obstinate, ill-tempered, abusive, and difficult to examine, but soon became complacent and even good-humoured, when conversing on the subject of his delusions. He said that he was the commander-in-chief, that the Queen was his mother, that he went with her in a yacht to Russia, to see his sister who was married to the Czar, that he, with forty comrades, killed 10,000 Russians at the Malakoff tower, and on the same day stripped the corpses, dug a hole, buried them, and sold their clothing for £20. He also boasted of his imaginary exploits in killing bears and tigers, and stated that he was General M—, had been forty years in the army, was about to present new colours to his regiment, was a district inspector and owner of the whole of Dover. There was considerable failure of memory. R Potassii iodid. and physostigma.

For some time after admission he showed great impatience, and often indulged in denunciatory language when not addressed by one of his assumed titles. Now and then he threatened violence when displeased, but gradually became more quiet in demeanour. The delusions of pride and ambition remaining still of the same nature, but varying in their expression from day to day.

Sept. 5th. The sight had gradually grown worse since his admission. Liq. strychnis m. iv—vi. to be injected beneath the skin of the mastoid or temporal regions each day.

Sept. 12th. Left pupil dilated, very sluggish. Right pupil contracted, almost immobile, and of irregular shape.

Sept. 29th. By the ophthalmoscope was seen a somewhat bright, white, atrophic pallor of the optic discs, with a somewhat irregular outline, and the vessels much diminished in size. Left pupil the larger, both of moderate size, irregular shape, and almost immobile. He was now nearly blind.

Nov. 2nd. The patient was stupid, heavy, confused, although able to reply, the face was flushed, the ears red and hot, the skin sallow, the temperature raised. He was in bed, but when supported upon his legs the left limb failed beneath him, and he kept pushing with his whole power towards the left side. R. K. Br. and K. I., elevation of, and cold to, the head.

Nov. 3rd. Still somewhat flushed. Feet, especially the left, rather cold. There was left hemiplegia, especially affecting the arm, the sensibility of which was also diminished: the face was scarcely affected. Pulse 84, and full. Temp., right axilla, 100°; left do., 99·4°. Pupils as on Sept. 29th (*suprd*). Relief of symptoms occurred after free action of enema of Mag. Sulph. and Turps.

Nov. 5th. The left hemiplegia was much less. Urine still drawn off by catheter. He was now in his more usual mental state, recounting to himself his various exalted notions with much satisfaction, but becoming foul and abusive in his language, on occasion. Nov. 6th. The right leg now appeared to be the weaker.

Nov. 10th. Left hemiplegia was again present, but was not very marked in the left upper limb, though the left leg failed greatly. Urine still removed by catheter.

Nov. 12th. The paralysis was again more marked in the left upper limb, and was well-marked in the lower, and only slightly in the face. The body, usually bent towards the left in standing, was occasionally bent towards the right. Nov. 20th. Seemed to be more helpless and stupid. Pulse 124; respiration 27; temperature 100·5. Signs of slight tubercle of left lung. 21st. Temp., right axilla, 101·5°; left do., 101·3°; pulse 105. Dec. 2nd. Had been placed on perchloride of iron.

After this he sat up every day throughout, his skin was of a dingy, coarse, muddy appearance. He muttered fragments of his old delusions; at other times he was querulous and morose, or abusive, or lachrymose. His selfishness and jealousy were childishly prominent, and now, as always, when angry he indulged in profanity and obscenity.

Jan. 11th, 1876. He expressed delusions as to matter lodging in his head, and running down over his whole body, especially over his back, and into his left leg.

Jan. 14th. Feet much swollen, ordered to bed.

Jan. 18th. Feeble, had diarrhœa, deglutition became very feeble, and his appearance drowsy and languid; he would scarcely reply. At night the hands and feet were cold; the pulse frequent, soft and feeble.

19th. This morning he was very heavy and stupid, apparently unconscious, but deglutition was fair and respiration as usual. The attendant said that suddenly at 8 A.M. he seemed worse, the breathing became laboured and quick, and then failed, and he seemed as if dying,—then he revived. A similar occurrence took place at 9 A.M., and the respiration soon afterwards was 50 per minute. At 10 A.M. the respiration was 36, irregular and loud, the pulse 116 and feeble, the frame was warm. The patient moaned, and was unconscious. There was paralysis of the right limbs and of the left third cranial nerve: the left pupil was moderately dilated, there was left ptosis, and the left eyeball, turned upwards and outwards, had lost the parallelism of its axis to that of the right globe. The head turned to the right, but was not fixedly so.

The coma, the rapidity of respiration, and the dysphagia increased. Respiration was mainly diaphragmatic. The tongue was dry, brown, and caked.

At 1 p.m. respiration was 57, loud, the pulse filiform and at times imperceptible. The palsies remained. He looked sallow, yellow, and thin, the skin was dry, rough, muddy in hue. The left pupil was wider than at 10 a.m., and both pupils were immobile. Temp., right axilla, 101.5°; left do., 101.3°.—Later in the day the breathing progressively augmented in rapidity and loudness. He remained quite comatose, and in the condition just described, until death, at 6.40 p.m., Jan. 19th, 1876.

Necropsy.—Forty-four hours after death. Calvarium of ordinary thickness, the portion removed, 14½ ozs. The dura-mater was slightly thickened. The left middle cerebral artery, and the two or three branches from it lying over the insula, contained an embolus half an inch in length, distending the vessel, of a dull pale reddish colour, elastic, friable, of granular fracture, and similar to that found in the pulmonary artery, especially its right branch.

The left posterior communicating artery was very small, the right posterior cerebral artery was smaller than its fellow, and the bifurcation of the basilar artery was displaced towards the left side. The walls of the vessels at the base were of normal thickness, but those of the right middle cerebral, and termination of the right carotid, contained a few opaque, yellowish white patches (atheroma).

Olfactory bulbs rather wasted. The optic nerves were softened and markedly atrophied.

The membranes, only moderately vascular, were thick, dense, and faintly opaque over the superior and lateral surfaces of the hemispheres, especially over the frontal lobes, and there, also, the pia-mater was infiltrated with serum.

Adhesions of the pia-mater to the cortex were present on the superior and lateral surfaces, principally over the posterior part of the frontal lobes, and to a less extent over the anterior and lower portions of the parietal lobe on the left side. There were scarcely any adhesions over the occipital lobe and the upper and posterior portions of the parietal. They were seen to a slight extent over the anterior two-thirds of the internal surface. The temporo-sphenoidal lobes were moderately affected thereby. The distribution of the adhesive change was much the same on the right side as on the left. The middle and anterior parts of the inferior cerebral surface were slightly affected. The angular gyrus, the postero-parietal lobule, and occipital lobes were almost entirely free from this change.

The convolutions were somewhat wasted; the sulci rather wide in the superior and external frontal-parietal regions.

The grey cortical substance of the cerebrum was rather pale, especially in the deeper layers, which were of a dull slightly yellowish hue and were firm, whereas the superficial layers were rather soft. About the vertex, where its deeper layers were of ordinary consistence, the left grey cortex was less wasted than the right, and in a section made at the level of the corpus callosum the grey matter of the left hemisphere seemed to be of greater depth in all regions, and was of an uniform pale grey colour. The right cortex was paler than the left, and its deeper layers were more uniformly hardened.

The white cerebral substance was considerably more vascular in the left hemisphere than in the right. Its consistence on both sides was somewhat diminished. Grey commissure large and soft; fornix soft. The lateral ventricles were rather large and contained a considerable amount of serum, their lining membrane was granulated, especially that of the right lateral ventricle. The veins of Galen contained more blood on the left than on the right side. About the middle of the ventricular aspect of the left corpus striatum, closely, but not immediately, beneath its lining membrane, was a small, softened, reddish patch, and a similar patch was seen in one of the orbital convolutions adjoining the insula. The left optic thalamus was of mottled hue, the right paler. The consistence of the basal ganglia was diminished.

Weight of right hemisphere, 18 ozs.; of left, 19½ ozs.

Cerebellum rather pale, and of slightly diminished consistence, 5½ ozs. Pons Varolii and medulla oblongata the same, and their weight ¾ oz. The lining membrane of the fourth ventricle was granulated and rough. About 13iii of serosity, in all, escaped from the cranial cavity.

Thorax.—*Heart*, 10½ozs. Milky spot on heart.

Some reddish granular clot, like that in the brain, was attached to the interstices of the right ventricle. Similar clot of a granular kind was found in the pulmonary artery, especially in its right branch. Some thickening of the edge of the anterior flap of the mitral valve. Muscular substance of heart of a dull pale and mottled hue. The left coronary artery had a whitish thickened patch on its inner surface (incipient atheroma).

Left Lung, 17½ozs., shrunken. Old general pleuritic adhesions, puckered cicatrices, and two or three encysted chalky masses of fair size at the apex and several smaller ones. Surrounding these were fibroid induration and a number of dirty whitish granulations, and the lung had here a dull greenish-grey colour. Stellate thickening of posterior surface of pleura.

Right Lung, no adhesions, 18½ozs., slight congestion.

Spleen, 6½ozs. A thick, irregular patch on the anterior and upper surface of the capsule, about two inches square, and of fibro-cartilaginous density.

Kidneys, surrounded by adventitious membranes and thickened capsules. Left kidney 6ozs. Right 5½ozs. Both in a state of suppurative nephritis with pyelitis, and embolisms.

Liver, 59½ozs., pale, flabby.

A little muco-purulent fluid in the bladder, and a small soft creamy collection in its wall, at the summit.

Microscopical Examination.—The embolic mass filled a portion of the left middle cerebral artery and of some of its branches, like a closely fitting cork. Like that in the right pulmonary artery, it had under the microscope an appearance of fibrillation, together with numerous leucocytes, and oval and irregular-shaped cells containing dark molecules.

The softened portion of the left corpus striatum exhibited blood corpuscles, red and white, and compound granule masses.

Of the optic nerves the right was apparently the smaller, both were atrophied and softened. The nerve tubes broke down under the glass, and fat globules, granule masses, and free molecules were seen.

Heart. There was slight incipient granular fatty degeneration of the muscular substance of the heart.

Liver. The hepatic cells were rather small, many of their angles unduly rounded. They were highly fatty, and there was much free extra-cellular fat, also.

Kidneys. The soft swollen portions with little whitish masses showed altered elements, lymphoid cells, molecular debris, and fat granules.

Remarks.—1. In this case amaurosis occurred at an early period, and gradually became complete. The blindness thus found at times in general paralysis has been attributed by some to double lesion of the angular gyri. But in this case the angular gyri were amongst the parts least affected by disease in the superior and external surfaces of the cerebrum. In certain other cases of general paralysis, also, the blindness is dependent upon a lesion which begins in the optic nerves themselves and their peripheral expansions, and not upon primary disease of the angular gyri, or of any part of the brain proper.

2. The apoplecticiform and paralytic seizures with left hemiplegia, recurring for several weeks and months before death, had evident relationship to the more marked disease of the right cerebral hemisphere.

3. During the last 30 hours of life there were marked right hemiplegia and paralysis of the left third cranial nerve, coming on suddenly, and associated with coma. The description given in the history of the case justified the provisional conclusion that some sudden lesion, probably pressure or invasion by blood clot from meningeal hemorrhage, had involved the left crus cerebri, although the state of the temperature was adverse to this view. But the necropsy showed embolism of the left middle cerebral artery, and embolic red softening of part of the lenticular nucleus of the left corpus striatum, as well as general greater injection of the left white substance and basal ganglia than of the right.

These conditions readily account for the sudden palsy of the right limbs and face, but how came about the palsy of the left third cranial nerve?

The irregular distribution of the arteries at the base of the brain has been described, and in looking for points of difference between the left and right third cranial nerves, one could only note that the left was contained within the hook formed by the termination of the basilar artery, deflected to the left, and the commencement of the posterior cerebral artery. The left cavernous sinus contained a soft dark clot; the right, none. One was almost constrained to think, that when the embolism produced sudden local stoppage and disorder of circulation that the distended and unusually-placed arteries may have compressed the left third nerve. Or, might one adopt the view of Brown-Séquard and say that the paralysis was due to the reflex local inhibitory effect exercised by lesion of some part of the encephalon?

4. The cerebral embolus was apparently of the same origin as the pulmonary embolus, and there arose the question of its origin in *débris* swept into the circulation from the kidney.

5. The kidneys exhibited destructive nephritis from the retrogressive irritation (!) of retained and decomposed urine, the atony of the bladder having been decided.

CASE XVII.—*Attack insidious in onset, protracted in duration. Exalted delusions, complacency, expression of generous and benevolent designs. Later on, in the middle periods, bien être, high opinion of self, but exaltation and exalted delusions not so marked as before, and mingled with some grumbling; and the speech less impaired. Later on, the patient sometimes confused, stupid, dazed, sometimes excited and irritable, usually morose, querrulous, sullen, grumbling, his language at one time most foul and obscene, and at another expressing extreme discontent, and, occasionally, delusions of annoyance, or of a hypochondriacal nature. Then, still later, some complacency mingled with, and alternated with, this last condition, and, finally, all decided expression of feeling was erased.*

The motor signs of general paralysis ordinary, irregularly, and slowly progressive; 4, 6, 8, and 11 months before death temporary "paralytic" seizures of left hemiplegia. Ten weeks before death, apoplectiform and epileptiform seizures, followed by coma, variable right hemiplegia, and Cheyne-Stokes' respiration; return of left hemiplegia, limbs, especially the left, somewhat rigid, and usually extended. Acute herpes.

Right hemisphere 2½ozs. less in weight than left, and its grey cortex more atrophied and paler and firmer,—slightly too firm in anterior regions. Right white substance decidedly indurated, especially in front. Similar, but less marked and extensive, induration in left hemisphere (grey and white). Adhesions, more marked over right hemisphere, and principally over the middle tract of the 1st, 2nd, and 3rd frontal g., over the orbital g., supramarginal and second temporo-sphenoidal g. The meningeal changes very highly marked and extensive, found in less degree over cerebellum and spinal cord. Ventricles large, ependyma thickened and opaque. Basal ganglia alike on two sides, pons and med. obl. rather firm and pale, spinal cord softened, pale, degenerated.

H. B., Private 4th battalion Rifle Brigade. Admitted May 29th, 1874, then aged 25 years. Service, 1½ years, single.

History.—This attack of mental disease was said to be the first, and of uncertain duration. He had been previously under treatment in 1873 and 4 in India, then on voyage home, and at Netley. "Predisposition and tropical climate" were the causes assigned for the attack, which came on in an insidious manner.

When he was admitted at Netley, on April 27th, 1874, complacency and exalted delusions were evident, and the articulatory power was affected as in general paralysis. He was incoherent in conversation, and gave expression to various exalted delusions, saying that he was a "general officer," and had "enormous wealth."

The principal points to note thus far were the insidious origin of the disease; and

its protracted duration at the time he was first sent to my care as an ordinary general paralytic.

State on admission. Physical.—Of a broad frame, and rather stout, muscles large but flabby, fresh complexion, pupils of natural size, susceptible, of vertically oval shape. Tongue protruded slightly to the right. Facial expression heavy. The speech was thick, and there were tremors and twitchings of the face, lips, and tongue, when in action; these tremors were slightly more marked on the left side of the face. Pulse 98, respiration 18. Slightly suspicious signs at the apex of the right lung. In the groin was the scar of a bubo, which he said he had at the age of 18. He denied having ever contracted syphilis.

Mental State.—He had extravagant and exalted delusions, saying that he was a general, a field-marshal, and had £12,000 a day. "Is as strong as a lion, and as sound as a church bell." "Can speak two Indian languages, and has a dozen pair of socks." Thus he propounded a sort of anti-climax—a descent from the magnificent to the trivial. He was self-satisfied, complacent, and expressed himself in a generous and benevolent manner.

July 22nd. Weight 175lbs., pupils as on admission, speech markedly impaired, the face and lips tremulous or twitching, especially during speech. The patient declared that he was well and strong, and he had high notions about himself. He was respectful to the officials, and patronizing towards the other patients. He was taking perchloride of iron.

Feb. 1875. He was now highly self-satisfied, and declared that he never had been insane. The memory was fair for simple matters, but the power of attention was lessened, he could not proceed easily from one subject of discussion to another, and in enforcing his views became loud and somewhat declamatory, and then one could observe the muscles of the face and lips tremble much and twitch, and there was a hesitating pause in the speech, which at times rose to a decided stammer, with clipping of the words. When he was perfectly calm, however, the speech was better than it was formerly.

May 19th. He had been confused, stupid, and dull for two or three days. May 22nd. About the same; ears and face red and heated, pulse usually about 100 and sharp. R. Aperient enemata, podophyllin, warm baths, and cold to the head. Under this he improved, but on May 24th, he was again dull and heavy, wore a sour sullen expression, would scarcely reply to any question, and said "they are killing me."

June 11th. Better since, but on this day was thinner, looked dull, unhappy and depressed, and said that "he felt badly." He shook tremulously when standing, and was unsteady in gait. There was ocular mucous hypersecretion. Oleum morrhue and liq. arsenicalis were added to the mixture of perchloride of iron which he was now still taking, although for a time he had been on K.Br. and digitalis.

Oct. 7th. The facial expression was heavy, dull, and rather sulky. He would reply to questions, but was averse from conversation. At times he now had strange delusions, such as that he was about to be buried in the w.c. He would purposely dirty any article of clothing of which he disapproved, was often obstinate, refusing to parade, and would not occupy or amuse himself in any way, but grumbled and growled morosely, first about one thing, then about another.

March 9th, 1876. Had been very peevish, morose, irritable, bad-tempered, swearing, grumbling, and using foul language, for many months; but notwithstanding this he occasionally said he "was all right," "there was nothing the matter with him," "he was as hearty as any man." Suspicious signs were heard at the apices of the lungs.

March 28th. Was now passing through an attack of acute herpes zoster over the front, inside, and partly the outside, of the left thigh, with some pemphigus blebs. The herpes extended upwards from the groin, trending outwards above the crest of the ileum to the parts overlying the sacrum and lower lumbar vertebrae. There was an isolated patch of herpes over the inside of the head of the tibia. No complaint was made of pain. This eruption left cicatricial traces.

May 23rd. During my temporary absence he passed through an attack of

left hemiplegia, which was said to have occurred without convulsion or loss of consciousness, and to have disappeared in about two weeks.

In July, his general health was improved, and he was taking a large amount of exercise. He was often restless, noisy and irritable, abusive and foul in language.

Sept. 23rd. Apparently he had had a paralytic seizure during the night, for upon rising in the morning he was found to have left hemiplegia, which grew less decided in a few hours. The right pupil was somewhat dilated, the left rather small, and both were sluggish. Sensori-motor reflex action was much less to impressions on the left than on the right eye, and the sight of the left eye was bad. He wept and chattered to himself, and expressed delusions of injury.

Nov. 10th. At 4 A.M. his night-attendant found that he had left hemiplegia again, but this diminished during the day, the paralyzed thoracic limb being flexed, the pelvic limb extended, and both rigidly resistant to passive motion. Temperature, right axilla, 97.8° ; left, 99° . There was not nor, as far as known, had there been any loss of consciousness. The tongue was protruded slightly to the right side; no very decided paralysis was observed in the face. The ears were injected, the face was of usual hue.

Nov. 11th. He was regaining power in the left limbs. Yet the palsy was detectible in the face. Temp., right axilla, 97.2° ; left ditto, 97.9° . He was childish and amnesic.

Jan. 5th, 1877. Had been exercising regularly since shortly after the date of the last note, was delighted with his power to do so, and repeated for hours together his favourite phrase "I'm fit to walk about." He talked good-humouredly as a rule, but occasionally had been noisy and obstinate.

This morning he fell down suddenly, and, upon being picked up, was found to be drawn to the left side, and to have lost power more or less in the left limbs and left side of the face. In a short time this palsy diminished, and the tongue was protruded slightly to the right, its tip inclining to the left. Temp., right axilla, 97.3° ; left, 97.8° .

Jan. 11th. He was now able to walk, but there was still some trace of the left hemiplegia.

March 16th. He had continued to be up and about from the date of the preceding note until this day, when, after appearing for several days to be flushed in the face, he suddenly sank down from his seat and became unconscious for a short time, after which he was quiet, looked dazed, could not reply to questions, was unable to swallow, but vigorously resisted any passive motion. Then, at 11 P.M. he had three epileptiform seizures, beginning in the right hand which, also, was much shaken by movements executed at the shoulder and elbow joints, and the fingers, though shaken by the spasm, were held rigidly extended. The convulsions were followed by *right* hemiplegia, and an insensative state of the conjunctivæ, especially of the right. An enema of chloral hydrate was ordered, after which the patient fell into a heavy sleep, and the convulsions did not return.

March 17th. The right hemiplegia was now only slight. Pulse 114, full, soft, bounding. Respiration 25, of the "Cheyne-Stokes" character, the cycle being short, the period of apnoea occupying about one-third of each cycle. Later on, after coughing, the rhythm of this respiration became modified, and there were simply ascending and descending periods of greater and less frequency, depth, and loudness of respiration. Temp. 101.2° . The face was flushed; the right conjunctiva insensative; the left sensitive. There were some pneumonic patches in the lungs. The patient was being supported by nutritive enemata.

March 19th. The head was boring over to the left side, the eyes turning thitherward. The thoracic limbs were flexed. The attendant said he had had a general convulsion, beginning at the mouth, after which he was flushed. The pupils were equal, of medium size, sluggish, the right conjunctiva was now again almost insensative. The right upper extremity was flaccid, the left rigid, and there was conjugate deviation of the head and eyes to the left.

March 20th. Was swallowing a little food for the first time since the 15th (save a little on 18th), having been supported by nutritive enemata *interim*. A convulsion,

however, occurred again last night. Pulse 60. Temp., right axilla, 97.4°. The conjunctivæ were now equally sensitive again. The thoracic members, especially the left, were rather rigidly flexed. (Was having ol. morrh., ferri perchlor., and vin. rubr. and K. Br.)

April 8th. Bedridden of late, quiet, replied to simple questions. Face generally flushed. This day there was slight left hemiplegia, the left upper limb was rigidly flexed. No convulsions recently. There was much grinding of the teeth.

April 9th. Dull, heavy, stupid, the face congested, deglutition worse.

May 5th. Bedridden since, often grinding the teeth, and having marked flushings of the face and head. For some weeks he had swallowed very badly. There was sinistral hemiplegia, and the left arm was rigidly extended. The patient was wet and dirty, and indulged in foul and obscene denunciation of imagined personal persecutions.

May 8th. In much the same condition. There were gangrenous fætor of the breath and signs of hypostatic pneumonia; and pneumonic patches at the apex of the right lung in front. R Terebinth. May 12th. Bleb on right thenar eminence. May 15th. The palsy continued to be more marked in the left limbs, but was now well-marked in the right also. An acute bed sore had appeared over the right first metatarso-phalangeal articulation: ordinary bedsores were forming also. Temp., right axilla, 100.6°; pulse, 98. May 17th. Left limbs thoroughly palsied and slightly rigid this day. The right were less palsied. Slight left ptosis.

To summarize: after this the condition remained much the same until death on May 25th, the pulse and respiration becoming very rapid on the last day of life. The mental life and power of expression were much effaced. He changed colour often and suddenly on the last day of life, and died on May 25th, 1877.

Necropsy, twenty-four hours after death. Moderate emaciation.

Calvarium, portion removed 15½ ozs.

Brain.—Rather firm, pale, clot occupying the first half inch of the left middle cerebral artery, not occluding it, but apparently formed before death. Olfactory bulbs wasted; considerable interlobar adhesions, slight thickening and opacity of membranes over the base and internal surfaces of the hemispheres. The pia-mater and arachnoid covering the superior and external surfaces of the cerebrum were pale, thick, and somewhat opaque, these changes being especially marked in the frontal and parietal regions. The membranes were infiltrated with serosity over the frontal and parietal regions.

Adhesion of membranes to the cortex was rather extensive, and was somewhat more marked over the right than the left hemisphere, and the usual decortication of the summits of the gyri resulted.

On the right hemisphere it was extremely well-marked over the greater part of the orbital surface, especially the first orbital gyrus; over the middle and anterior part of the first frontal gyrus, but absent at its tip; over isolated spots at the same level on the second frontal gyrus; on the anterior part of the third frontal, avoiding the tip; over the supra-marginal and second temporo-sphenoidal gyri.

It was pretty well, but very much less, marked over the temporo-sphenoidal lobe generally, the rest of the parietal lobe not already mentioned, and the lower and anterior part of the occipital lobe.

The adhesive change was but slight on the surface adjoining the superior longitudinal fissure on this hemisphere, though it was found in a few scattered spots here, but the upper part of the internal surface, just within the fissure, was affected slightly, and more so in this than in the left hemisphere.

In the left hemisphere this change was best marked on the central portions of the orbital surface, over the anterior part of the first frontal and second frontal gyri, but avoiding the tip, and over the posterior part of the third frontal gyrus, the rest of which was intact.

It was present, but in a very much less degree, over the posterior part of the left second frontal gyrus, encroaching upon the middle of the surface of the ascending frontal gyrus; and affecting the upper extremity of the ascending parietal gyrus, the postero-parietal lobule, and the supra-marginal very slightly.

It was very faintly seen scattered over the whole length of the second temporo-sphenoidal convolution, while the rest of the temporo-sphenoidal lobe, the rest of the parietal, and the whole of the occipital lobe were intact in the left,—in this respect differing from the right.

The grey cortical matter was rather thin, the convolutions generally were slightly wasted and pale, though in parts exhibiting a few dilated vessels. Its consistence was perhaps slightly increased, especially in the fronto-parietal region. The grey matter was slightly paler, and was thinner, in the right than in the left hemisphere.

The white substance, especially that adjoining the cortical grey matter of the frontal lobes, was of somewhat increased consistence, and it was pale.

In the left hemisphere, the increased consistence of the grey and white substances diminished backwards, being normal in the posterior third of the brain. In the right hemisphere the increase of consistence was more marked, and was at its acme in the white substance of the frontal lobe, which on section presented a slightly cribriform appearance, and was at this part of a pure brilliant white colour. Thence the induration diminished backwards, through the parietal and temporal lobes into the occipital.

The veins on the walls of the right lateral ventricle were fuller than those of the left. The ventricular lining membrane was thickened and opaque, and the ventricular cavities were enlarged, and contained considerable serosity. In appearance the two corpora striata were almost alike. The basal ganglia were not plump.

Weight of right cerebral hemisphere $15\frac{1}{4}$ ozs. ; of left, $17\frac{1}{8}$ ozs.—the difference, $2\frac{3}{8}$ ozs.

The pons Varolii and medulla oblongata were rather pale and of slightly increased consistence. Weight, $1\frac{1}{2}$ oz. The lining membrane of the 4th ventricle was thickened and opaque.

Cerebellum. Meninges slightly opaque ; grey matter rather pale ; consistence fair ; weight $5\frac{1}{2}$ ozs. Serosity, $5\frac{1}{2}$ ozs.

The spinal cord was rather pale and soft. On microscopical examination, hæmatoidin pigment masses were seen on the surface of the cord, by the meninges ; and colloid bodies—among other changes—were widely scattered throughout its substance.

Thorax.—*Heart*, $9\frac{1}{2}$ ozs., its muscular substance pale and friable, valves healthy, a few patches of atheroma in the coronary arteries, and at the commencement of the arch of the aorta.

Left lung, $37\frac{1}{2}$ ozs. *Right*, $39\frac{1}{2}$ ozs. ; lobular and hypostatic pneumonia and semi-gangrenous patches in both lungs.

Right Kidney, $7\frac{1}{2}$ ozs. ; *left kidney*, $6\frac{1}{2}$ ozs. *Spleen*, $4\frac{1}{2}$ ozs., rather soft. *Liver*, 50 ozs., of pale pinkish hue, capsule thickened and adherent on upper surface.

Remarks.—1. Connected with the much more advanced disease of the right hemisphere were, in all probability, the attacks of left hemiplegia which recurred on several occasions,—finally returning to an extreme degree before death, and accompanied by rigidity of the left limbs.

2. It is not easy to assign the exact cause of the apoplectiform seizures, followed by epileptiform convulsions, coma, *right* hemiplegia, with some anæsthesia, and Cheyne-Stokes' respiration, ten weeks before death. This right hemiplegia fluctuated and returned incompletely before death.

3. Acute herpes zoster affected the left thigh, left buttock, and lumbo-sacral region fourteen months before death ; latterly there were blebs on the right hand and foot, occurring in a rapid manner ; these were contemporaneous with rigidity of the left limbs, which were in a state of extension. These conditions might depend upon irritative sclerotic lesions, especially affecting the left side of the spinal cord, both the internal radicular fasciculi of the posterior columns, and the antero-lateral columns.

So far have the intended limits of space been overpassed that merely the headings of the remaining cases will be given, together with some of the appended remarks.

GROUP V. (see p. 183).

CASE XVIII.—*Excitement, discursive conversation, exalted delusions, emotional exaltation and joy, alternating with anger and hostility, hallucinations of hearing(!), patient, quarrelsome, abusive, and foul-mouthed. Later on, the exalted delusions continued, were associated with much maniacal restless violence, and insomnia, and a boastful cynical disposition. The language was more and more profane, obscene, and abusive, and the habits became wet and dirty. Frequent attacks of stupor or semi-stupor, in which he resisted passive motion, but swallowed well, and sometimes with these were spasmodic tremors of the head, trunk, and limbs, and arms at times rigid. Between these he was very noisy and restless at night. Many, and frequently recurring, epileptiform convulsions, at first not followed, but in the later stages followed, by unconsciousness, and afterwards by a character of speech and of mentation often observed in epilepsy. These convulsions occurred singly, or in bouts, and latterly were very severe and frequent, and associated with marked stupor and local spasms of the trunk and left limbs. Finally, rigid flexed contractures of all the limbs, and continuance of epileptiform seizures. Throughout, the impairment of speech well-marked, an unusual amount of stuttering, very great motor restlessness, and marked tremor of the muscular system.*

In the right frontal lobe, the grey cortex of the superior and external surfaces was markedly indurated, and was atrophied, of a red colour, and hypervascular. This condition was mainly limited to this lobe, the rest of the right grey cortex being only slightly firm, and far more normal in appearance, as was, also, the whole of the grey cortex of the left hemisphere. White substance, injected in right frontal lobe, pale in rest of right hemisphere, of ordinary vascularity in left; and universally of equal and slightly increased consistence. Adhesion and decortication considerable, especially over parietal lobes, also well-marked over frontal lobes. Lateral ventricles, especially right, large; their ependyma firm. Right basal ganglia pale, and right corpus striatum somewhat shrunken. Pons and med. obl. rather firm, their meninges thickened. Spinal cord somewhat softened in its upper portions, and the left posterior cornu atrophied in dorsal region.

Details as to meningo-cerebral adhesions. In the right hemisphere there were a few points of adhesion on the prominences of the ascending convolutions, especially of the ascending frontal; also on the posterior part of the inner surface of the first frontal. The third frontal was affected. The external surface of the temporo-sphenoidal suffered considerably. The inferior surfaces of the temporo-sphenoidal and of the frontal lobe were comparatively much freer from this change. In the left hemisphere, as compared with the corresponding portions of the right, the anterior two-thirds of the parietal gyri suffered much more, the superior surface of the frontal gyri suffered a little more, and the external surface of the temporo-sphenoidal considerably less, than the corresponding parts on the right side. The left third frontal convolution was quite free. The following were the parts most affected by this change, in the order of severity,—left parietal; right parietal; right temporo-sphenoidal; left temporo-sphenoidal; left frontal; and right frontal lobes.

Microscopical examination.—*Fresh tissues.* Grey cortex of right first frontal convolution. Much neuroglia; hyperplasia of neuroglial nuclei and of the vascular, with hypertrophy, also, of the latter. Some vessel-walls irregular and rather thick. Many small round and oval-cells. Pyramidal nerve-cells small. In the left first frontal convolution the nerve-cells and vessels were more normal. In the fresh spinal cord the multiplication of nuclei and condition of cells were much the same.

Prepared sections.—In the right first frontal gyrus these did not take the carmine stain well, and there were unstained patches of a ground-glass appearance. The walls of the vessels were thick. The nerve-cells were atrophied, some were granular, some of ground-glass appearance, some disintegrating. Round and oval nucleated cells; scattered dark pigment grains.

The left first frontal gyrus stained better than the right; its changes were the

same as in the right, but they were considerably less marked. Of the right third frontal the same may be said, except that it more nearly approached the condition of the right first frontal. A number of unstained sclerotic patches were seen here also. The left third frontal gyrus resembled the right, except that the nerve-cells were more granular. The spinal cord did not stain very well. The walls of some of its vessels were thickened and their nuclei hyperplastic; there was increase of the connective tissue; some aggregations of granules, and a few scattered compound granule masses were observed,—also numerous small round and oval cells containing either a nucleus or molecules.

Remarks.—1. As to the relation of the changes in the cortex of the right frontal lobe, or, again, the relation of the unequally and irregularly distributed adhesive changes,—to the localized spasms and convulsions, it is probable that, of the two, the convulsions were rather in relation with the change involving atrophy and induration of the cortex of the right frontal lobe. Whatever the relation of the morbid process causing cerebro-meningeal adhesion might have been to the condition of brain conducing to convulsion, it had no obvious bearing upon the localization of the convulsions and spasms observed.

2. The left third frontal gyrus was free from adhesion; the right was affected thereby. The sclerosed state of the cortex was probably connected with the affection of speech in this case. The speech was of the character which Voisin assigns to lesions of the cortical grey matter of the anterior convolutions, or, sometimes, the fibres passing thence to the medulla oblongata.

3. There were frequent attacks of stupor, either alone, or with spasmodic twitching of both sides of head, trunk, and limbs. It was a matter of question whether these attacks of stupor were due to active cerebral congestion, or to epileptic cerebral exhaustion, or temporary arterial occlusion.

4. The mentation at times assumed, in part, the characteristics of that often associated with epilepsy. The special type of noisiness, following the attacks of stupor with spasms, was of this nature; in several points there was a coincidence with epileptic delirium.

5. The question of syphilis of course arose in this case, and several of the necroscopical details might readily be deemed the traces of ancient syphilis. I refer to the conditions of the basilar artery and of the spleen; both, however, were slight and equivocal. The attacks of stupor were not unlike what sometimes occur with syphilitic disease of the cerebral arteries. Microscopically, the small arteries were noted as thick-walled.

6. Coincidentally, were atrophy of left posterior grey cornu of dorsal region of spinal cord, and long disuse of the left leg following upon a rare injury:—*transverse fracture of the patella from direct violence.*

CASE XIX.—Failure of mental powers, progressing to fatuity. No connected conversation. Smiling, contented, obedient, docile, quiet. Later, some restlessness and resistance to manipulation, habits wet and dirty. At an early period insensitive to discomfort; later, insensitive even to a painful operation,—quite analgesic apparently. Occasionally, stupid heavy drowsy states independently of the occurrence of convulsions. Impaired speech and co-ordinating power. Slight right hemiplegia. Right—and then left—and again right-sided hemispasm. Increase of paresis, especially in the right limbs. Twitching of the right upper limb, and choreiform movements supervening. Later, much right and left hemispasm. The right upper limb occasionally rigid, and then less resistant to passive motion. Occasional attacks of hemiplegia. Drowsiness, coma, slow and feeble pulse, low temperature, death.

Slight induration of the cerebral grey cortex in the superior and external fronto-parietal region, gradually lessening below and behind these parts. Grey cortex also atrophied, especially in the frontal lobes, the atrophy diminishing thence to tips of occipital lobes. Somewhat lessened vascularity, perhaps in the grey, certainly in the

white, substance, and in the meninges. Adhesion and decortication comparatively slight, and somewhat more in the right than in the left hemisphere, and mainly seen at the summit of the fissures of Rolando. Meningeal changes highly marked and extensive, and especially seen over parietal lobes. White substance slightly too firm; lateral ventricles large, ependyma thickened, as also in fourth ventricle. Basal ganglia somewhat pale and shrunken, pons and medulla oblongata slightly too firm.

On the *left* side the cerebro-meningeal adhesions occurred at the summits of the gyri where the first frontal, ascending frontal, and ascending parietal nearly meet, by the longitudinal fissure. On the *right* side, besides the ones corresponding to these, there were a few more adhesions affecting the posterior half of the upper surface of the first frontal gyrus, the posterior half-inch of the second frontal, the upper fourth of the ascending parietal, and part of the postero-parietal lobule close to the longitudinal fissure.

Remarks.—1. As to the history of syphilis, we note here the absence of lesions distinctly syphilitic, after death, and the absence of syphilitic symptoms during the period the patient was under my care.

2. In a case presenting so many motor symptoms it was of interest to find the adhesion and decortication within the so-called "cortical motor zone." Attributing the convulsive and spasmodic symptoms to the adhesive lesion, which was mainly about the summits of the fissures of Rolando, the facts would support the localization views of Carville and Duret—to a less extent those of Charcot and Pitres. But why the spasm and convulsion should have predominated on the right side of the body was a difficulty in the way of this hypothesis.

CASE XX.—Hypochondriacal and melancholic delusions, at first with gloomy depression, later with garrulous incoherence. Patient moaning and lachrymose, and, later, repeating parrot-like the words used by others, exhibiting fear, apprehension, and anxiety, and attempting to commit suicide; but subsequently becoming far less distressed. Early hallucinations of hearing, and, later, of sight. Finally, very incoherent and demented.

The ordinary motor signs developed very late indeed, long after decided mental disease. Patient finally became helpless, bedridden, and very wet and dirty; the limbs flexed and rigid. Habitual partial disuse, or stationary paresis, of right 7th and 9th cranial nerves. Left ophthalmoma. Left acute bed sore.

Some induration of the cerebral grey cortex in the superior and external fronto-parietal regions, gradually shading off backwards and downwards, to the normal consistence at the tip of the occipital lobe, and at the base. Grey cortex generally, pale, but streaked by visible vessels. White substance generally, of slightly increased consistence, and pale, especially in front. Meningeal changes ordinary, with considerable oedema. No adhesions of the soft meninges to the brain. Lateral ventricles rather large, fornix firm; spinal cord slightly softened.

Microscopical Examination.—First and second right frontal convolutions near the tip. Many small round or oval cells containing several dark molecules. The nuclei of the vessel-walls somewhat increased in number, and an increased number of "Deiter's cells." Slight atrophy and degeneration of some of the nerve-cells, several of which were partly surrounded by vacuoles.

Upper part of ascending gyri. Some of the nerve-cells of somewhat rounded outlines. Increased number and size of "Deiter's cells."

Tip of occipital. Many of the small round and oval cells above-mentioned.

Spinal cord, less changed than was anticipated. Some of the multipolar cells were undergoing degeneration. A few spindle-shaped cells with large nuclei were seen.

Remarks.—1. The case was peculiar in this, that the physical signs of general paralysis came on in a patient already more than a year and a half insane. Not-

withstanding the statements of the earlier writers on the subject, I deem this to be of singularly rare occurrence, and dependent upon the special primary localization of the morbid process in the cerebrum.

2. In this case we note, also, the *absence* of any adhesion of the meninges to the cerebral surface ; of any marked changes in the dura-mater ; and of any encephalic softening.

And, on the other hand, the *presence* of slight diffused hardening of the grey cortex in the supero-external fronto-parietal regions.

3. There was an absence of unequivocal syphilitic lesions after death, unless, indeed, the incipient aortic endoarteritis deformans be deemed a syphilitic lesion.

4. Acute (or hyperacute) bed sore (decubitus acutus) appeared on the left buttock forty-five days before death, the position and course of which corresponded with those of bedsores sometimes following lesions of the right cerebral hemisphere—while yet there was no circumscribed lesion of that hemisphere, and the morbid appearances were symmetrical in the two hemispheres.

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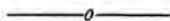
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